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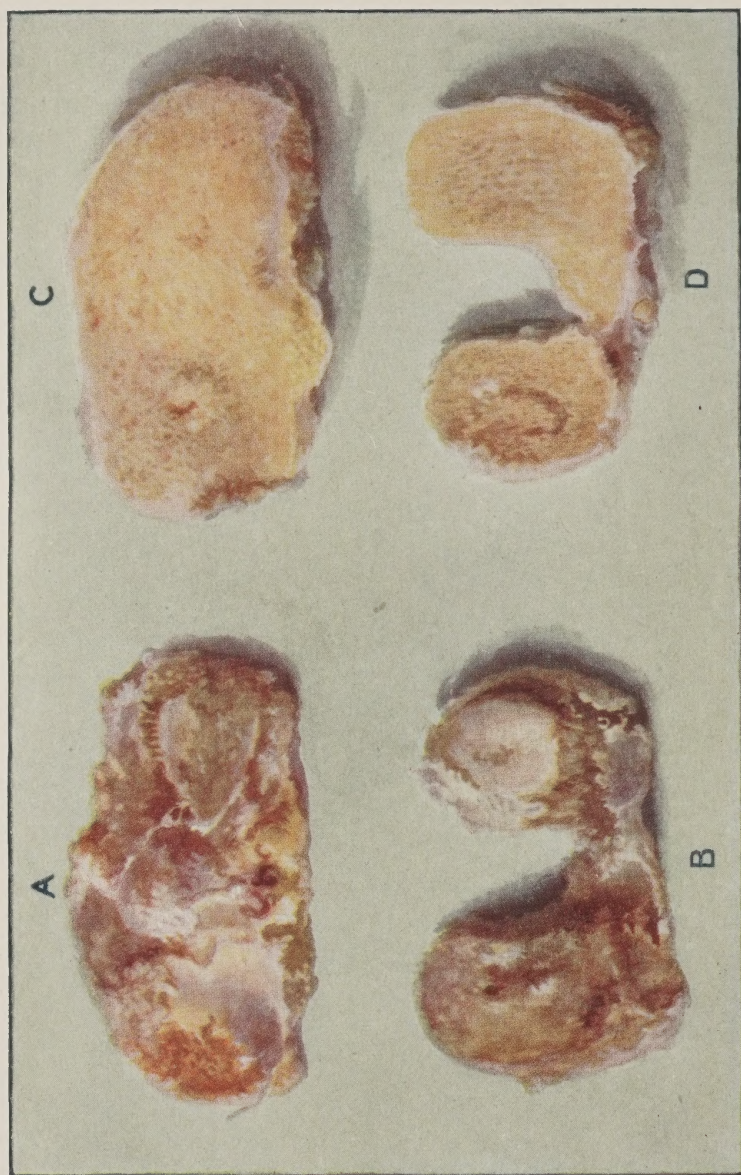
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Tuberculosis of Knee. Resection, with removal of about one centimeter from the end of femur and tibia.
 (A). Joint surface of tibia.
 (B). Joint surface of femur.
 (C). Cut surface of resected piece of tibia.
 (D). Cut surface of resected piece of femur. Note sequestrum.

INFLAMMATION IN BONES AND JOINTS

BY

LEONARD W. ELY, M.D.

ASSOCIATE PROFESSOR OF SURGERY, STANFORD UNIVERSITY

144 ILLUSTRATIONS



PHILADELPHIA AND LONDON
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TO MY WIFE

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PREFACE

TO WRITE a book on inflammation in bones and joints requires considerable temerity, and, perhaps, if I disown any undue self-confidence, I shall disarm criticism in advance. Unlike many subjects in medicine, great difference of opinion exists as to the physiology and pathology of these two organs. We have abundant clinical material on which to draw, but conclusions based on clinical opinion are notoriously conflicting. Like a religious argument, and perhaps for the same reason, an argument between the exponents of diverse clinical views is usually characterized by positive statement and by some acrimony.

We have at our disposal also the results of considerable investigative work, but much of this is not as well known as it should be, and the conclusions of the investigators do not always agree. Too often the laboratory worker does not take the trouble to check up his results by clinical evidence, and too often the clinical expert is unwilling to devote the time in the laboratory which is necessary if he would understand the fundamental processes in the tissues with which he deals. Without this knowledge he is simply guessing as to what lies beneath the surface.

When all is said, the gap in our knowledge is due to our ignorance of bone pathology, and we shall probably not fill it up until bones and joints are sectioned at necropsy with the same care as are other organs. We may not always agree with an observer's conclusions, but we all know, when we are searching through a mass of contradictory opinions, with what satisfaction we greet the publication of concrete, definite facts discovered in the examination of pathological material. We can draw our own conclusions from these facts.

Whatever this book lacks must be charged to the limitations of its author. It is based upon personal observation and research, and is not to be regarded as a compilation, though on numerous occasions I have taken pains to set forth the opinions of others where they differ from mine. The chief merit, I think, is its exposition of the results of original research, and of work in the pathological laboratory, and the correlation of this work with clinical findings. The facts unearthed in this way should be of value to others, even when the conclusions drawn from them are not accepted.

My colleagues in the Stanford Medical School, by their coöperation and encouragement have aided me in many ways, particularly Dr. Frank Blaisdell, Associate Professor of Surgery, in my animal work and in the illustrations. I am under great obligation to the librarians of the Lane Library, for their unremitting work upon the bibliographies, which should be of the greatest help to the student. The devoted work of my technician, Miss Wallach is beyond all praise.

The facilities which Stanford University puts at the disposal of its faculty are unique. As tools it furnishes a great library, laboratories, clinics and hospitals, all grouped closely, and grants the leisure to use them. To its president and trustees I make my respectful acknowledgments.

I take pleasure in acknowledging my indebtedness to Messrs. William Wood & Company of New York, to the Surgery Publishing Company of New York, and to the editors of *The Annals of Surgery*, the *Archives of Surgery* and the *Journal of the American Medical Association* for permission to reproduce material which has appeared in their publications.

San Francisco, California, 1923.

The Author.

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SECTION I.
GENERAL CONSIDERATIONS

INFLAMMATION IN BONES AND JOINTS

CHAPTER I.

GENERAL CONSIDERATIONS.

DISEASE in bone derives its special interest from the fact that it runs its course locked up in a narrow case or shell which influences its manifestations and treatment. Diseases of the small bones of the extremities, and of the ends of the long bones, are peculiar also in that they have in their immediate vicinity a closed cavity, the joint, whose involvement often overshadows the disease in the bone, and gives the clinical picture its stamp.

In our studies six tissues require examination: (1) Bone tissue proper, (2) Marrow, (3) Periosteum, (4) Cartilage, (5) Synovial membrane, (6) Ligament. Inasmuch as difference of opinion often springs from difference of definition, we shall define these terms as well as describe the tissues they represent.

Bone is an animal substance composed of organic material impregnated with salts of lime and magnesium.¹ Bone cells also are an integral constituent. Bone is divided into two classes: dense, and spongy or cancellous, but the bone in each is the same, and only differs in its arrangement and in its amount.

Dense bone is found at the circumference of the shafts of the long bones, and as a layer on the outside of all spongy

¹ Bone ash consists of calcium phosphate 84 per cent., magnesium phosphate 1 per cent., other calcium salts 7.5 per cent., carbonic acid 5.5 per cent.

bones or portions of bone. Under the articular cartilage it is prolonged as a thin layer in the normal state. On the inside of the cortex of the shaft it may end abruptly or it may be lined by a small amount of spongy bone. The second is the more frequent condition.

As it approaches the end of a long bone the layer of dense bone tapers off, to be prolonged under the cartilage.

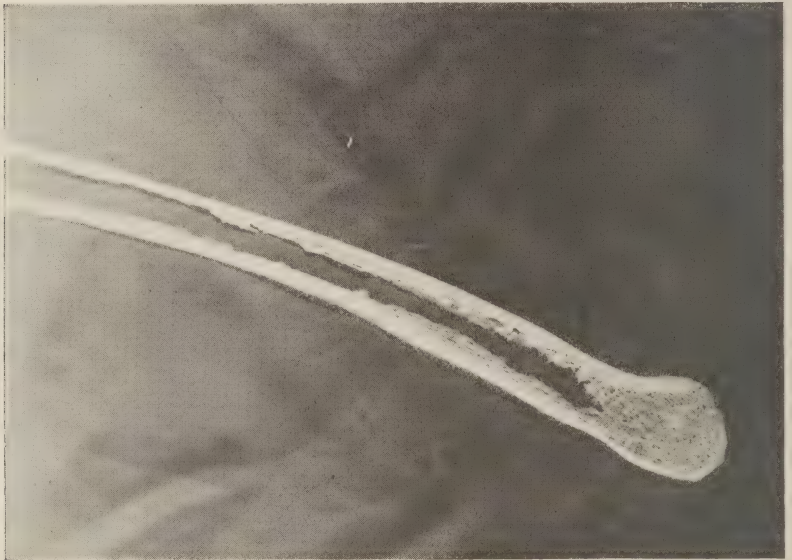


FIG. 1.—Slice of the distal portion of the normal human femur. Note the spongy bone on the inside of the cortex.

The inside of the cortex presents no analogue to the periosteum on the outside. There is no such structure as an “endosteum.” Endosteum is simply marrow.

Except under the cartilage the dense bone of the cortex is perforated by canals which transport blood vessels. Fibres of the periosteum also pass into it. In other words the inside of the bone is only relatively a closed cavity.

Spongy or cancellous bone makes up the bulk of the ends of the long bones, and the bulk of the short and the

flat bones, the vertebrae, the sternum, the ribs, carpus, tarsus, etc.

Bone tissue itself is not subject to inflammation, nor actively to disease, and simply reacts to disease or change in its contained marrow. Usually a mild irritation in the marrow causes an hypertrophy of the bone, a stronger irritation an atrophy. If the irritation in the marrow be



FIG. 2.—Low power photomicrograph showing blood vessel entering the bone cortex. Note absence of any such thing as endosteum.

very severe, the bone dies. We are accustomed to speak of diseases of bone. It would be more accurate to speak of diseases *in* bone.

Constant change is going on in the bone, that is, its structure is always changing to adapt itself to the uses to which it is put. If a part be put at rest, its bony structure atrophies, if much work is demanded the bone hypertrophies, but both processes are purely passive and are accom-

plished by the blood vessels in the marrow and in the periosteum.

Bone has two great functions. The first is its mechanical function as the framework of the body. This interests us especially in our study of deformities and of fractures. The second is as the container of the marrow. This is important in its relation to infection.

Bone can be injured in only two ways. It can be attacked, as we shall see, by disease in its contained marrow, or it can be fractured. It cannot be sprained, strained, or suffer contusion. This seems self-evident, and yet one often hears bone disease ascribed to the late effects of a bone injury, other than fracture. Such a thing is of course impossible.

MARROW is the soft tissue within the bone—all the soft tissue within the bone. Its situation, not its composition, determines its name. Wherever there is bone there is marrow, in the central canal and in the spongy bone, and even, to a small extent, in the dense bone itself. Tissue with all the characteristics of marrow is occasionally seen in the region of the joints, on the outside of the cortex, immediately under the fibrous periosteum.

Human marrow is of three kinds; first, red or cellular or lymphoid; second, yellow or fatty; and third, myxomatous or fibrous. The structure of marrow is so diverse and changeable that it is often difficult to say what is normal and what is not. Marrow consists of a reticulum of connective tissue, blood vessels, blood spaces or channels, fat, and a greater or smaller number of many kinds of cells. The proportion of fat and of these diverse cells determines the quality of the marrow, whether it is fatty or lymphoid. In myxomatous or fibrous marrow little is present but fibrous tissue.

The composition of marrow varies not only in health and disease, but also with age.² In the human infant practically all marrow is lymphoid. In childhood the lymphoid marrow gradually disappears from the shafts, and then slowly with the lapse of years from the ends of the bones also. The adult has little lymphoid marrow

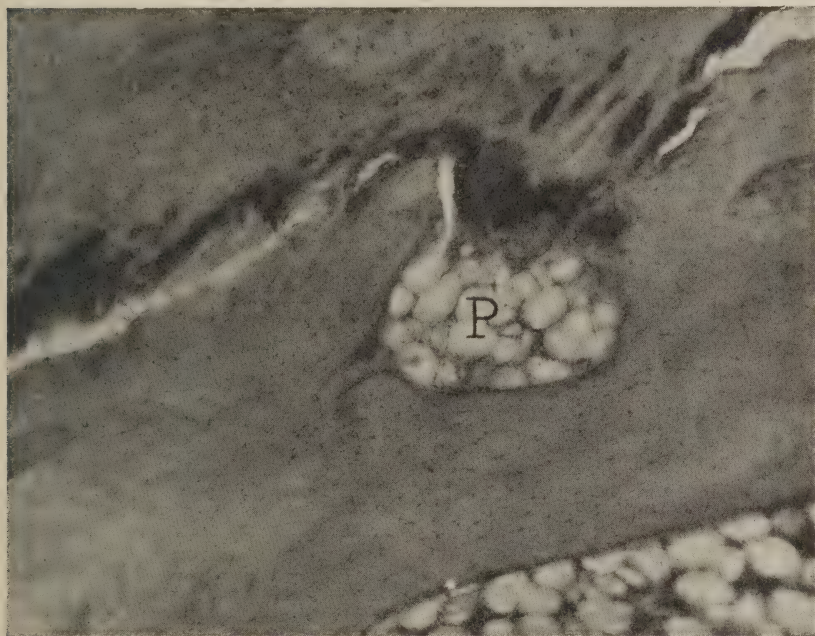


FIG. 3.—Marrow pocket on the outside of the cortex. Low power photomicrograph.

in his long bones. It persists longer in the bodies of the vertebrae.

In studying diseases in bone, the changes in the bone tissue itself first attract our attention, but give us very little information. Whether we examine the bone with the naked eye, with the microscope or with the Röntgen

² DICKSON, W. E. CARNEGIE: "The Bone Marrow." London, Longmans, Green & Co. 1908.

rays we observe that the changes in it are of the simplest kind, and are only three in number,—absorption or atrophy, production or hypertrophy, and death or necrosis. The farther we investigate, the stronger grows our conviction that all changes of bone tissue are purely passive,³ and are simply the result of changes in the contained marrow.

The marrow is one of the most complex, changeable, active and interesting tissues of the body, and reacts quite promptly to most infections. Fraenkel⁴ found typhoid bacilli in the bone marrow of every one of 110 patients dying of typhoid. Areas of necrosis in the marrow are frequently observed in autopsies of patients who have died of infectious diseases.

In laboratory animals dying of an infection, I have often noticed a marked engorgement of the marrow; so often that I have come to regard it as a pathognomonic sign of an infection. Probably the “aching bones” at the beginning of an infectious disease are due to this congestion. As we shall see later, the marrow in tuberculosis shows typical tubercles, in suppurative osteomyelitis, engorgement, pus cells, colonies of bacteria, etc., in chronic bone disease the fibrous changes later to be described.

The theory of the purely passive rôle of the bone tissue itself is not by any means generally accepted. In fact, it must be said that by far the weight of opinion lies on the other side. Most writers believe that the bone tissue itself is capable of active inflammation, and attempt to differentiate *ostitis* from *osteomyelitis*. In the same way the differentiation is made between *periostitis* and inflammation of the marrow (or bone) immediately below the

³ KLEMM, P.: “Die Osteomyelitis des Kindesalters.” Berlin, 1914. S. Karger.

⁴ FRAENKEL, EUG.: “Ueber Knochenmark und Infektionskrankheiten,” *Münchener medicinische Wochenschrift*, 1920, xlix, 561.

periosteum. Klemm, in his book on osteomyelitis, discards completely the terms *ostitis* and *periostitis*. Probably the periosteum, like any other fibrous tissue is subject to inflammation, but I think that the term *periostitis* as ordinarily employed is a misnomer, and that what we know as *periostitis* is really an inflammation of the subjacent bone



FIG. 4.—Low power photomicrograph of diffuse tuberculosis in the bone marrow.

marrow. The terms *rarefying* *ostitis* and *productive* *ostitis* are used synonymous with bone absorption and bone production, but seem to possess no particular merit.

The marrow, especially the lymphoid marrow, is to be regarded as a chemical laboratory, the bone simply as the building which houses it. A knowledge of the composition of the marrow gives us a ready comprehension of its vulnerability to infections and to their location as well.

The marrow of spongy bone, at least well into adult life, and in children the marrow of the shafts also, is essentially a lymphoid tissue. If pus germs of sufficient virulence are carried in the blood stream to a lymph node, the lymph node suppurates and breaks down; if they are carried to the bone marrow, the bone marrow does the same. The difference in the course of the disease in the two cases is, in the first place a question of the amount of tissue involved, and in the second place one of environment. In osteomyelitis the products of suppuration are shut up in an almost impermeable shell and this fact makes the process much more severe, and increases the danger immeasurably.

This explanation of the frequency of the occurrence of infections of the bone marrow in the young is not the standard one. Most authorities emphasize the ætiological importance of trauma. Some affirm that rapid growth predisposes to infection. Lexer⁵ ascribes to the arrangement of the blood vessels about the epiphyseal line, the causal rôle in the frequency of infections near the end of the bone. His view might be said to be at present the accepted one. The matter will be taken from the realm of speculation when the bones are sectioned at necropsy with the same thoroughness as the other organs of the body.

THE PERIOSTEUM is the tissue which covers the bone in all places except those covered by the joint cartilage. Its structure and function have been the subject of much discussion, into which we shall not enter at any length here. The difference of opinion is due partly to a lack of exact definition, and partly to faulty reasoning. A knowledge of the subject is advisable if one would understand the subject of bone formation.

⁵ LEXER, E.: "Weitere Untersuchungen über Knochenarterien und ihre Bedeutung für krankhafte Vorgänge," *Arch. f. klin. Chir.*, 1904, lxxiii, 481.

If one looks at the periosteum of the shaft of a growing bone, that is, of a bone of a child, one will often distinguish two layers, an outer or fibrous, and an inner or cellular layer. The cells in the latter are probably osteoblasts or bone-forming cells, such as are seen on the margin of all growing bone; such as are seen on the margin of bone

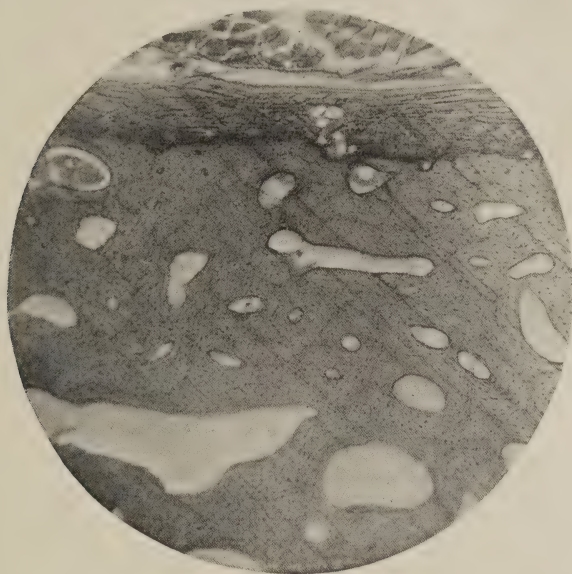


FIG. 5.—Low power photomicrograph of normal human bone, showing periosteum and fairly dense bone. The section was taken of the so-called metaphysis, that is in the region where the dense bone is changing to spongy. In this stained slide the bone cells appear as small dots.

trabeculae in the interior, when they are growing in thickness. These cells are described as part of the periosteum, but they really are part of the bone, and have nothing whatever to do with the periosteum proper. In the specimen from an adult this cellular layer may be present or absent.

The periosteum consists of a rather loose-meshed fibrous tissue. Here and there this fibrous tissue may be

replaced by cartilage, or by fibrocartilage. This is periosteum none the less, no matter what its structure.

Fibres of the periosteum run down into the bone, but, of course, as soon as they enter the bone they cease to be periosteum, and become marrow. With both marrow and periosteum the situation, not the structure, determines the name.

The blood vessels of the periosteum send branches into the bone. These run in small canals, and anastomose with the vessels of the marrow.

LIGAMENTS are composed of dense white fibrous tissue, and serve to bind the bones of an articulation together. Their fibres pass into the articulating bones, but some of the more superficial are continuous with the periosteum.

A JOINT is usually a closed cavity between two or more bones, and is bounded by two tissues, the synovial membrane and the cartilage.

CARTILAGE consists of cells and basement substance. The cells have a definite capsule and a characteristic appearance which often helps to identify the tissue under the microscope. The basement substance consists of collagen fibrils impregnated with chondromucin. Cartilage contains according to age 3 to 6 per cent. of mineral substances, of which calcium sulphate forms from 48 to 92 per cent.

The articular cartilage is of the hyaline type. Its basement substance appears homogeneous in the normal subject, but in certain joint diseases it loses this homogeneous appearance and takes on a distinct fibrous structure. The fibres run parallel to the surface superficially, but deeper in, at right angles to it. This fibrillar appearance

it said to be present also in joints which have been immobilized for any length of time.

The joint cartilage in the foetus is covered by a perichondrium, but in infancy, after function has been established in the joint, the perichondrium disappears. In certain diseases thereafter the superficial portion of the cartilage may take on the appearance of a perichondrium, especially about the margin where it shades into the synovial membrane, but the normal joint cartilage in the adult is without perichondrium.

The dense, smooth nature of the joint cartilage adapts it admirably for its functions of motion and of weight bearing, and these functions are subserved by the absence from it of blood vessels and of nerves.

It is well to remember that in childhood the joint cartilage is simply a portion of the cartilaginous epiphysis. As time goes on the growth of the bone nucleus separates it from the epiphyseal cartilage, until, with the disappearance of this, the articular cartilage becomes the sole remnant of the mass of hyaline cartilage of which the whole bone was originally composed. Cartilage may really be viewed as essentially an embryonal tissue doomed eventually to



FIG. 6.—Photograph of stained slide from a sagittal section of the knee of a normal dog.

ossification. Some of it ossifies early, some of it late. The individual constitution influences the time of ossification, as does the manner of life, and possibly, the occupation. Viewed in this light the stiffening of the joints as age advances, is easily understood. On the other hand some persons have supple joints which work smoothly even at an advanced age.

Fibrocartilage is found in the joints in the shape of interarticular discs or menisci. They serve to lessen shock or to add security. In structure they partake of the nature of the ligament and of hyaline cartilage. In the vertebral joints they add motion and stability. The joints of the vertebral bodies possess no hyaline cartilage, no joint cavity and no synovial membrane.

The articular cartilage possesses no blood vessels. No lymph vessels have ever been found in it, though their existence has been assumed by some authorities. Exactly how the cartilage is nourished is not known. It is supposed to draw its nourishment from the marrow beneath, and possibly from the synovial membrane at its margin. Certainly its whole reaction to disease seems to depend upon the condition of these tissues. It is not subject to inflammation, nor directly to disease. It simply suffers secondarily from involvement of the synovial membrane and of the marrow. Probably there is no such thing as a primary disease of the articular cartilage nor invasion of it from the joint side; authorities differ on this last point, the majority holding the contrary opinion.

The articular cartilage, and the epiphyseal cartilage while it is present, form a barrier to the spread of infection, practically an absolute one. The broad general rule may

be laid down that an infectious process can not make its way through a cartilage whose nutrition is intact. Hence infectious processes in the bone marrow, in order to reach the joint, must either travel around the margin of the cartilage, or else first shut off its nutrition from beneath, and then perforate it. If the view is correct, as I believe it is, that the cartilage is immune from invasion from the



FIG. 7.—Section of dog's patella, camera lucida drawing. Joint cartilage below. Note the little pouch at either end of the cartilage, lined with synovial membrane, to allow for motion. Note also the buttress of bone beneath the cartilage, and the absence of any perichondrium.

joint side, then any infection, to pass from the joint to the bone marrow must travel around the margin of the cartilage.

THE SYNOVIAL MEMBRANE is a connective tissue structure which bounds the joint in all places except those bounded by the articular cartilage. It covers intra-articular ligaments, and, in certain joints, notably the knee, is prolonged inward in the form of fringes or curtains. It secretes a viscid fluid which lubricates the joint, and normally is just sufficient to serve this purpose and no

more. The membrane is continuous with that lining certain of the bursæ in the immediate neighborhood of some joints, so that disease in one readily spreads to the other. There is, of course, no such thing as a hernia of a joint.

The normal synovial membrane is smooth and shining, but in the region of its junction with the cartilage, it is thrown into folds, to accommodate it to the movement of the bones. Here its cells have much the appearance of epithelium.

The junction of the cartilage and the synovial membrane is not abrupt, but the two tissues shade gradually into each other, so that it is not possible to say exactly where one stops and the other begins.

When a joint is immobilized for any reason, the synovial membrane encroaches upon the cartilage at its periphery, replaces it, so to speak, and extends especially over its surface. When motion is restored, provided the joint has not been damaged by disease, the cartilage extends again at its periphery, and regains its former limits.⁶ This is probably the cause of the stiffness of joints after immobilization. It is doubtful if any adhesions, properly speaking, are ever caused in a normal joint by immobilization.

The reaction of the synovial membrane to injury or to disease is very interesting. If it be irritated it pours out a

⁶ NATHAN, P. W.: "The joint cartilage in its relation to joint pathology." *Am. J. of Orth. Surg.* 1909-10, vii, 85.

BRANN, HEINRICH: "Untersuchungen ueber Bänder Synovialmembranen und Gelenkknorpel." *Deut. Zeit. f. Chir.*, 1894, xxxix, 35.

REUBER, CARL: "On the cartilages and synovial membranes of the joints," *Jour. Anat. and Physiol.* 1874, viii, 261.

HAMMAR, J. AUG.: "Ueber den feineren Bau der Gelenke." *Archiv. f. Mik. Anat.*, 1894, xliii, 266, 813.

secretion whose nature depends, of course, upon the irritant. The mere presence of this fluid probably causes thickening and inflammation of the membrane. It is thrown into folds, and takes on a villous structure. These villi may attain great size and number, giving to the inner surface of the joint a marked shaggy appearance.

No stomata ever have been demonstrated in the membrane, and the method of the subsequent exit of the fluid from the joint is not known. Strange to say, fluid does not accumulate in the joints in cases of œdema of the extremities.

Any infection of the synovial membrane causes the thickening and villous condition, and the characteristics of certain infections are

beautifully shown in the membrane, as they are in the bone marrow.

The opinion formerly prevailed that the synovial membrane was a closed sac extending out over the cartilage, but this is not a fact. It is a section of a tube, ending at the border of the joint cartilage, with which, as has been said, it is continuous. In the embryo, however, and for a

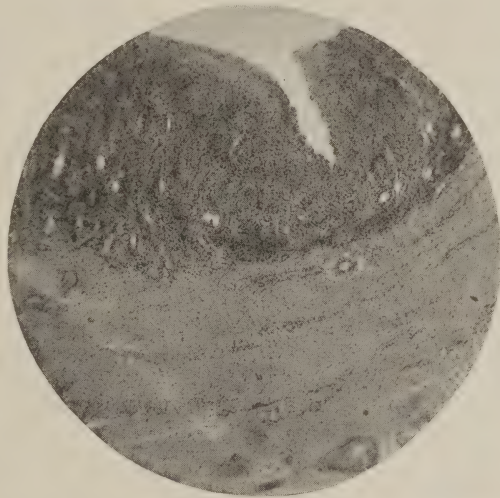


FIG. 8.—Low power photomicrograph, showing typical appearance of the synovial membrane in an acute inflammation. This is from a case of probable syphilitic arthritis wrongly diagnosed as tuberculosis and infected at operation. Synovial membrane above. Ligament below.

while after birth, the synovial membrane is continuous with the perichondrium.

In all joint inflammations the synovial membrane is to be regarded as the active tissue, the cartilage as the passive. It follows that the terms synovitis and arthritis are synonymous. With an arthritis, the adjacent bone marrow may or may not be involved; that is, an osteomyelitis may or may not be associated with the arthritis.

We recognize, then, in inflammations of bones and joints, only two active tissues; the bone marrow and the synovial membrane. The ligament, the cartilage, the bone tissue itself, and probably the fibrous periosteum all play a purely passive rôle. We are wont to use the term "periostitis" quite frequently, but the real inflammation in such a case is usually in the bone marrow in the immediate vicinity.

Broadly speaking the marrow and the synovial membrane are vulnerable to the same infectious agents. Any bacteria which invade one can invade the other. However, certain infections show a preference for the marrow, certain for the synovial membrane, while certain others affect both tissues without preference. A knowledge of this fact helps us in our diagnosis. Thus *treponema pallidum* belongs in the first class, the *gonococcus* in the second, and the *tubercle bacillus* in the third.

We note also that while some infections always start in the lymphoid marrow, and practically always remain there, others start in either the lymphoid or the fatty marrow, with about equal frequency. The lymphoid marrow in the ends of the long bones seems to be the great chemical laboratory in which most infections germinate; that in the

short bones to a lesser degree. We shall see how they may never travel beyond the limits of this tissue in certain instances. Indeed, as in the lungs, only the necropsy may reveal that they have ever been present at all. In most cases, however, they show a tendency to spread.

The direction in which the various infections spread is also a matter of interest, and often an aid in diagnosis. Tuberculosis always travels toward the joint if it travels at all. Pus infections usually travel toward the shaft, less often towards the joint, but streptococcic infections show the contrary tendency, and travel toward the joint.

Fundamentally the pathological characteristics of all bacterial bone and joint infections are the same, as are the characteristics of flowers or of trees. We distinguish among them by their minor traits. One great difference exists between the botanist's task and ours. He has his object exposed to his senses, while ours is covered by the skin and subcutaneous tissues. Not until we have a culture of the offending organism are we sure of its identity, but the more carefully we study its habits, its life history, so to speak, and its effects, the more often shall we be able to recognize it well enough for all practical purposes, and to institute our measures of cure, without waiting for a positive identification. On the other hand, remembering the impossibility of reaching a positive conclusion without the aid of the microscope, we do not overestimate our diagnostic ability, and consequently do not so often make humiliating mistakes.

THE FORMATION OF BONE, 7, 8, 9, 10, 11, 12, 13

The phenomena of bone production have been known for a long time, but the identity of the active agents of the process, and their exact rôle, are still a subject of discussion.

Until comparatively recently the "metablastic" theory of bone formation prevailed. According to this the various members of the connective tissue group possessed the power in certain circumstances of changing to one another. This theory is at present not widely held. It has generally been displaced by the "neoblastic" theory, which predicates the existence of a definite bone-forming cell, or, "osteoblast."

The exact identity of the osteoblast is not known. It is described as a small, round or polyhedral cell, with sharply staining nucleus, usually seen on the borders of the trabeculæ or on the outside of the cortex, but without characteristics clearly enough defined to permit its identification away from its accustomed habitat. We see these cells in great numbers on the outside of young growing bone, and on the edge of the trabeculæ when we believe that bone is being built up, and we are wont to conclude, there-

⁷ AREY, L.: "The Origin, Growth and Fate of Osteoblasts," *Amer. J. of Anatomy*, 1920, xxvi, 315.

⁸ TODD, T. W.: "Development and Growth of Bone," *Journal of Anatomy and Physiology*, 1912-1913, xlvii, 177.

⁹ BUSCH: "Die Osteoblasttheorie auf normalen und pathologischen Gebiete," *Deutsche Zeitschrift für Chirurgie*, 1878, x, 59.

¹⁰ BILROTH: "Anatomische Beobachtungen über das normale Knochenwachsthum," *Archiv für klinische Chirurgie*, 1864, vi, 712; "Ueber Knochenresorption," *Archiv für klinische Chirurgie*, 1862, ii, 118.

¹¹ MEYER, ARTHUR: "Side Lights on Multiple Myeloma," *American Journal of Medical Sciences*, 1918, clvi, 329.

¹² ELY, LEONARD W.: "The Formation of Bone," *Ann. of Surg.*, 1919, lxi, 225.

¹³ LESER, EDMUND: "Ueber die histologischen Vorgaenge auf der Ossifications Grenze," *Archiv f. klinische Chir.*, 188, xxxvii, 511.

fore, when we see these small cells in this situation that bone formation is going forward. This is not always true. The borders of the trabeculae often present the same appearance when we know quite well that the bone is being torn down. This has led some observers to the conclusion that

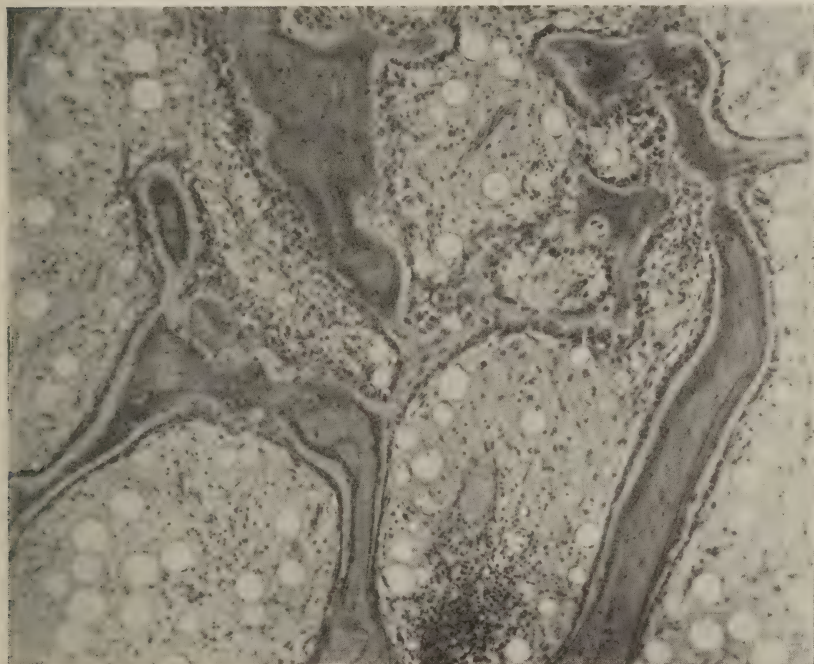


FIG. 9.—Low power photomicrograph from a case of marrow tuberculosis. The bone is being torn down, not built up, but the edges of the trabeculae are covered with cells corresponding to the ordinary description of osteoblasts. Note the interval between these cells and the trabecula.

the same cell which builds bone up, can also tear it down, that is, that the osteoblast is the same as the osteoclast. In this view I am inclined to concur. I have in my possession stained slides of marrow of tuberculosis and of acute osteomyelitis, in which all the evidence points to bone absorption, but in which cells with the typical appearance

of osteoblasts are seen in great numbers on the borders of the trabeculae.

The prevailing view is that the osteoclast is a large cell—a giant cell—seen usually in an excavation on the border of the trabecula (Howship's lacuna) or at a short distance from it. Some observers believe that this giant cell is the

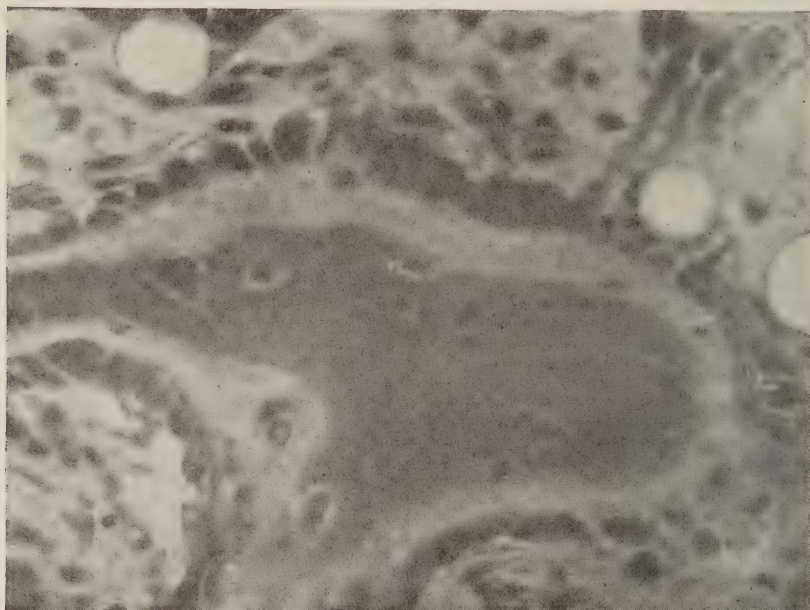


FIG. 10.—High power photomicrograph of a portion of the preceding slide.

result of the bone absorption, and not its cause. Bone probably can be absorbed without the agency of any special cell, by simple absorption of its lime salts—"halisteresis."

Whence comes the osteoclast, if there be such a cell, is not known. The origin of the osteoblast is still unsettled. Geddes¹⁴ says it wanders in from the epiblast, but it is generally considered a mesoblastic cell. Some say that it

¹⁴ GEDDES, A. C.: "Origin of the Osteoblast and Osteoclast," *Journal of Anatomy and Physiology*, 1912, xlvii, 159.

is brought by the blood stream, but Moschcowitz¹⁵ says that it is simply a mesothelial cell arising in the process of angiogenesis. He says that the endothelial cell in the vessel wall, the osteoblast, the osteoclast and the bone cell are all fundamentally the same, with the same potentialities.

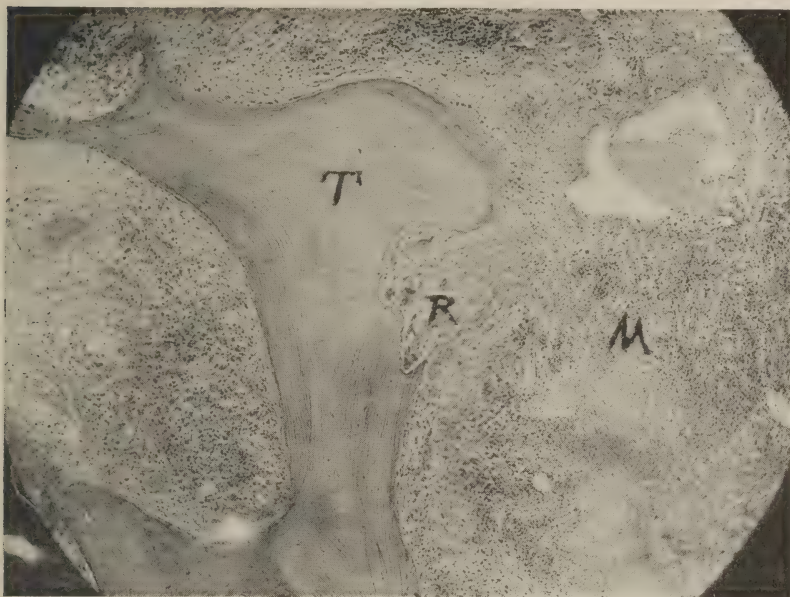


FIG. 11.—Low power photomicrograph from a case of acute suppurative arthritis. T, trabecula, M, marrow. Most of the trabecula is dead, but its upper left, and its lower right hand border show signs of a deposit of new bone. The bone cells here stain. The marrow is the seat of an intense inflammation. At R appears what is known as rarefying osteitis.

What function they eventually possess simply depends upon their surroundings.

Stained slides from specimens of fractures or resections, or from the inside of the cortex of growing bone, where nature is rearranging the architecture of the bone, show the classical giant cell osteoclast in great numbers. When,

¹⁵ MOSCHCOWITZ, ELI: "The Relation of Angiogenesis to Ossification," *Johns Hopkins Hospital Bulletin*, 1916, xxvii, 71.

however, the bone is being absorbed as the result of an infectious process in the marrow, or after being buried experimentally in the soft tissues, these giant cell osteoclasts are few in number or are absent altogether.

If the neoblastic theory is correct, as we shall assume that it is, then three things are necessary for bone formation, and these are: first, the active agent, the builder; second, the building material; and third, the stimulus.

Ollier,¹⁶ in 1867, first described a "cambium" layer of the periosteum and ascribed to it a bone forming function. The question was debated for years, until finally the periosteum came to be regarded as the great active agent in bone formation. In 1912 MacEwen¹⁷ denied this function, and affirmed that the periosteum was simply a limiting membrane and that the marrow formed bone. The question is still being debated, and much experimental and clinical evidence has been adduced on both sides.¹⁸ Without going into the details of the controversy, let us assemble the known facts concerning bone formation, and see if we cannot reach a satisfactory conclusion and one that will be of practical service. Manifestly we cannot throw out the evidence submitted by reliable observers, and yet we cannot accept conclusions diametrically opposed. The chief difficulty disappears when we remember that neither the periosteum nor the marrow forms bone, but the osteoblast.

¹⁶ OLLIER: "Le Régénération des Os," 1867, v, Masson et fils, Paris.

¹⁷ MAC EWEN, WILLIAM: "Growth of Bone, The," 1912, Maclehorse and Sons, Glasgow. The Macmillian Co., New York. 1912.

¹⁸ HASS, S. L.: "Regeneration of bone from periosteum," *Surg. Gyn. and Obst.*, 1913, xvii, 174.

JOKOI: "Experimentelle Beitrag zur Knochenneubildung durch Injections bez. Implantation von Periostemulsion," *Deutsche Zeitschrift für Chirurgie*, 1912, cxviii, 433.

The following statements will hardly meet with opposition:

1. Intracartilaginous bone formation begins with the pushing of blood vessels into the cylinder of cartilage in a long bone. Around these blood vessels calcification and ossification take place. A similar process takes place later



FIG. 12.—The blood vessel has pushed its way into the epiphyseal cartilage, and has initiated bone formation in the centre of ossification. Low power photomicrograph from the knee joint of a foetus at term. New bone in upper left hand corner.

in the epiphysis. This is bone formation without marrow or periosteum.

2. Bone is formed in the walls of the aorta, and in the kidneys of laboratory animals whose renal vessels have been tied off.¹⁹ Again bone formation without marrow or periosteum.

3. Bone is formed from cartilage or from fibrous

¹⁹ LIEK: "Zur Frage der heteroplastischen Knochenbildung," *Archiv für klinische Chirurgie*, 1906, lxxx, 278.

tissue, in the marrow of adult bones, without the aid of the periosteum.

4. Bone is formed in fibrous tissue—intramembranous bone formation—before any marrow is present.



FIG. 13.—Low power photomicrograph of early stage of intracartilaginous bone formation. Blood vessels pushing their way into the cartilage.

5. Bone is formed on the outside of the cortex, beneath the periosteum, again bone formation without marrow.

6. New bone is sometimes formed in necrotic or hyaline tissue, *e.g.*, lymph nodes, tuberculous foci, corpora albicantia, thickened pleuræ, etc., etc.

7. Buried bone contacted with other bone and enjoying a functional use, persists and is renewed. Bone buried in the soft tissues, without function, slowly disappears.

8. Bone is never formed except in the presence of blood vessels. The entrance of blood vessels into a tissue to be ossified is the first step in the process.

If we sum up our facts we find that three things are necessary for bone formation:

1. Blood vessels—the builder.

2. Either (a) a loose meshed fibrous tissue, or (b) a homogeneous (cartilaginous matrix), or a granular or a necrotic material—the building material.

3. A stimulus, physiological or pathological as the case may be. It is this stimulus which causes the blood vessels to push into the cylinder of cartilage in the first place, and which causes the bone production in the aorta, for instance. We recognize function as a stimulus, and the mere presence of bone as another.

It is therefore seen that neither periosteum nor marrow is essential for bone production, and that neither of them forms bone, in the proper meaning of the word. In each tissue we simply conclude that the conditions are suitable for bone production. The materials are there, and given the stimulus, physiological or pathological, bone will be manufactured out of the fibrous or cartilaginous tissue in the periosteum or marrow. The true marrow cells, the characteristic marrow cells, the cells which give the marrow its stamp and its function, probably have no rôle whatever in bone building.

We observe other phenomena in the formation of bone, which are of use to us in our clinical work:

Cavities in bone are filled up to the old level, in the absence of infection. Normally the new bone will not go beyond the old level. The shape of the cavity does not materially influence this process. In the presence of a pus infection the shape of the cavity does influence it.

In such a case steep walled cavities will not fill in. The walls must be beveled off before healing will take place.

It is doubtful if nature ever fills up with new bone the cavities in bone caused by certain old closed chronic infections. Old cheesy tuberculous foci may persist for years. They may show calcification but not ossification. *They do not become vascularized.* Old bone cysts may persist indefinitely. On the other hand, bone destroyed by a syphilitic process, can probably be restored by nature without artificial aid.

The examination of old specimens of buried bone teaches us that sterile dead bone, under the stimulus of function, is replaced by nature. She employs the old bone as a scaffold. The details of this will be set forth under the head of bone transplantation.

Bone cannot grow out any distance from its own level, unless it have a bridge or scaffold on which to grow. If the head of the femur be cut off, a new head will not grow on the neck. If the shaft of the tibia be removed for the cure of a suppurative osteomyelitis, new bone grows out for a very short distance from the pieces of bone left behind, and then stops. Nature perhaps uses the periosteum as a scaffold and builds bone along it to connect up the fragments.

It may be laid down as a general rule that in bone, as in other tissues, a mild irritation, especially an intermittent one, causes an hypertrophy, a severe one, an atrophy. If the irritation be very severe, the bone dies—necrosis. Whatever the ultimate cause of the process may be, the immediate cause of the hypertrophy, the atrophy or the necrosis is to be sought, in the interior of the bone in the vessels of the bone marrow, at the surface in the vessels of the periosteum, and of the superficial cortical marrow.

HALISTERESIS.—Formerly the view was widely held that bone could be removed by the action of some ferment which first dissolved the lime salts without the medium of any special bone destroying cell, any osteoclast. One sees this view seldom now-a-days in American text-books, but there is much evidence to support it, and I believe it is true, in many instances, possibly in all.²⁰ In sections from malignant growths in bone, this halisteresis is often evident.

The dense bone of the shaft has less marrow and fewer blood vessels than has the spongy bone, and therefore we should expect to see it in less activity both in destruction and repair, than in the spongy bone or in the central marrow canal. In point of fact all the activity in the process of repair after a fracture, is observed at the surface of the cortex, as will appear hereafter.

THE HEALING OF FRACTURES

If a hole be bored through the cortex, new bone is not built straight across the gap, but bone trabeculae, springing from the inner aspect of the cortex, form in the marrow of the central canal. These slowly increase in number and in thickness until they plug up the hole and extend up into it. This is the so-called "internal callus." To a lesser extent the same process takes place on the outside of the cortex. Bone trabeculae spring from the exterior surface of the cortex, bridge the gap on the outside, extend down into the gap, and meet those coming up from the marrow. The resulting callus is beautifully likened by Nichols,²¹ of

²⁰ KAUFMANN, EDWARD: "Lehrbuch der speziellen pathologischen Anatomie," Berlin, G. Reimer, 1917.

²¹ The late DOCTOR EDWARD HALL NICHOLS had a most remarkable series of slides showing this process, and to him I am indebted for my knowledge of it. In his death the medical profession suffered an irreparable loss.

Boston, to a collar button, which it strongly resembles in its shape.

It is probable that, after a fracture, if the fragments could be brought into accurate apposition and could be held in absolute immobility, this same collar button would form, with a small head and base, and with an infinitely slender shank. In practice, however, this is not what we find. The continuity of the marrow is broken as well as that of the cortex. Nature seems to find great difficulty in building bone across the smallest gap, unless she have a scaffold or bridge on which to build it. She builds this bridge in the following manner:

Immediately after a fracture hemorrhage takes place into the marrow canal and on the outside of the cortex beneath the periosteum, which has already been stripped from the cortex by the fracture to a greater or less extent. The hemorrhagic exudate on the outside of the cortex is replaced by granulation tissue, and this in turn by fibrous tissue and cartilage. As time goes on then the cartilage is ossified by penetration of blood vessels. The greatest activity in this process is seen to be manifested on the external aspect of the cortex, and especially in the angle between the cortex and the periosteum. I have watched this process taking place in three series of experimental fractures on cats, and in no instance have I been able to detect any evidence that the periosteum takes an active part in it. The sole function of the periosteum is to hold the soft callus firmly against the bone until cartilage has formed in it.

This external callus is the chief means of joining the two bones together and may be compared to the "wiped joint" with which the plumber solders together two pipes. It holds the bone ends absolutely immobile until the union is complete, and until the external surface of the cortex of

one fragment is firmly united to that of the other. It probably takes about a year for the fractured fragments to be united directly, and for the normal bone architecture to be restored. Then, if the apposition have been absolutely exact, the callus presumably is removed entirely. The apposition probably never is absolutely exact; hence some



FIG. 14.—Low power photomicrograph from a stained slide taken from a section of the outside of the cortex of the tibia of a dog. The knee joint in the immediate vicinity had been resected fourteen days previously. Note the new trabeculae, T, T, T, T, forming on the outside of the cortex under the periosteum.

of the callus always remains, and betrays the site of the fracture.

The formation of this external callus may be seen in laboratory animals, and its development may be watched also with the Röntgen rays. The bone fragments are seen to be united by a firm bony bridge long before there is any evidence of union between the ends of the cortices. This

direct cortical union probably does not take place for about a year.

The so-called internal callus, so prominent when a hole is bored in the cortex of a laboratory animal, plays an insignificant rôle in the healing of an actual fracture. In my

experimental fractures it was usually rudimentary, and never bridged any gap between the two fragments. It certainly does not show in the X-ray plate. The bridge of bone from the external surface of the cortex of one fragment to that of the other seems to be the essential feature of the union. Where for any reason it does not form non-union is wont to result.

Nature seems to inaugurate two distinct and opposite processes after a fracture. With one hand she starts to build a bony bridge under the periosteum and in the mar-



FIG. 15.—Photograph of a stained slide from a sagittal section of the knee of a dog. The knee had been resected seventeen days previously. Large pieces of bone had been removed.

row, as described, with the other she tries to construct a joint. If one looks through the microscope at the ends of the bones of a laboratory animal some days after the production of a simple fracture, one sees that they are separated rather than joined by fibrous tissue which streams out from the marrow canal to the periosteum.

The same thing is seen after experimental resection of

the dog's knee joint.²² If non-union result, this fibrous tissue becomes greatly thickened, and increases in amount. Some of it runs across from side to side, some of it between the marrow and the periosteum. Cartilage cells often appear in it, and almost invariably clefts, which may or may not be lined with a synovial membrane. Eventually a new joint is established at the site of the resection, a new joint which bears a striking resemblance to that seen at the site of an old un-united fracture.

In the great majority of these experimental resections a new joint is formed. Bony ankylosis is extremely hard to secure. The exact reverse prevails after simple fractures in laboratory animals. Bony ankylosis is almost invariable, but bony union is very rare if the periosteum be divided circularly. If the periosteum be simply slit before the bone is fractured, union may or may not take place.

Non-union is very rarely seen in the child, in whom new bone formation under the periosteum is very active. It is said to occur often in certain constitutional diseases, notably syphilis. A favorite site of bone syphilis is immediately under the periosteum. Impacted fractures almost invari-



FIG. 16.—Photograph of stained slide from sagittal section of knee of dog, resected 374 days previously. Small pieces of bone had been removed, 6 mm. from condyles of femur, 4 mm. from head of tibia. Fibrous union with many small clefts in the fibrous tissue.

²² ELY, LEONARD W.: "Experimental resection of the dog's knee joint," *Annals of Surgery*, 1919, lxx, 586.

ably unite firmly if they are left alone. Non-union is notoriously frequent in portions of bone not provided with periosteum, such as the carpal bones and the head of the femur and the head of the radius. Lack of contact between the fragments tends to prevent union, as does the interposition of soft tissue.

Weighing all the evidence, clinical and experimental, we reach the following conclusions:

If the two fragments of a fractured bone not covered by periosteum can be brought into exact apposition, and can be held firmly together without motion, they will unite directly after a long time. If they be not held close together, or if any motion take place, a new joint probably will be formed.

The rôle of the periosteum in the healing of fractures is to hold the soft callus against the outside of the cortex. Where the periosteum is absent or



FIG. 17.—Photograph of stained slide of sagittal section of knee of dog, resected twenty-seven days previously. Small pieces of bone have been removed. The end of the femur had been sawn convex antero-posteriorly and the end of the tibia concave antero-posteriorly. The bones are tightly united by fibrous tissue.

divided, non-union is wont to occur.

Various other reasons have been advanced for the occurrence of non-union, notably deficient blood supply and alcoholism.

The opinion is quite widespread that fractures fail to

unite much more frequently than formerly, and for this the Röntgen rays have been held responsible. The work of Albee and Morrison²³ tends to disprove this. They found that exposure to the Röntgen rays did not retard the process of union after experimental fracture of rabbit's

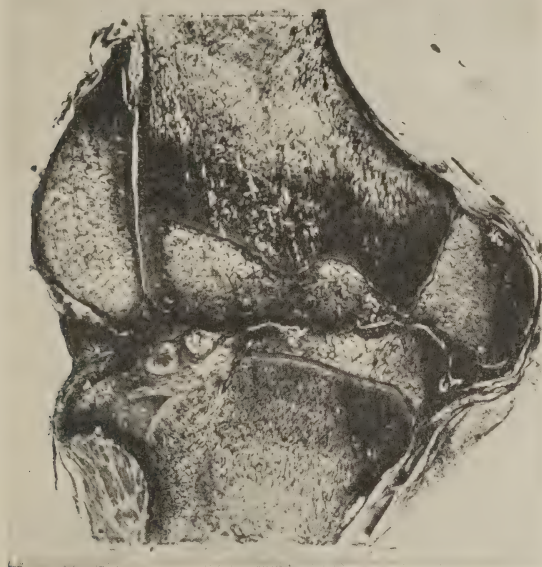


FIG. 18.—Photograph of a stained slide from a sagittal section of the knee of a dog, resected 150 days previously. Note the absence of bony union. The union is fibrous and in the fibrous union are large clefts.

bones. On the other hand, non-union is extremely rare in animals under any conditions. I have seen it only once in a simple fracture, but have produced it in two out of four open operations on cats, in which I divided the periosteum completely, and fractured the humerus. In another series of simple fractures, union was almost invariable, if the

²³ ALBEE AND MORRISON: "Studies in bone growth: an experimental attempt to produce pseudarthrosis," *Amer. Jour. Med. Sciences*, 1920, clix, 40.

animals were permitted to live long enough for the ordinary fracture to unite.²⁴

Besides the changes at the site of a false joint set forth above, there are others even more important from a clinical standpoint. If the bone ends are exposed to pressure, as



FIG. 19.—Photograph of a stained slide from a sagittal section of the knee of a dog. The knee had been resected 790 days previously. Moderately large pieces had been removed from the bone ends, and the ends were shaped in mortise fashion. Note that the bones are separated by a band of fibrous tissue, in which is a large cleft.

in the tibia after a simple fracture, new bone often is laid down at their periphery, so that they become broadened, like the ordinary epiphysis of a long bone. All the elements of a permanent joint are then present, including fibrocartilage over the bone ends, and a synovial membrane. The fibrous tissue between the two bones is dense and is incapable of conversion into bone. It separates the two marrow canals. It replaces the periosteum. Frequently also one or both marrow canals are shut off by a layer of bone, from this fibrous tissue.

Without artificial assistance no union is possible.

In other instances, especially after compound fracture, more or less absorption of the bone ends takes place. They become conical, very dense and hard, "eburnated," and

²⁴ Three series of operations were done. In the first the fracture was a simple one, by direct violence: In the second, the periosteum was slit longitudinally, and the bone was divided with Liston forceps: In the third, every effort was made to sever the periosteum in continuity, before the bone was divided. *Archives of Surgery*, 1922, v. 527.

contain little soft tissue except a few blood vessels. Their ends are bound together with dense fibrous tissue. Two bones whose ends are eburnated will never unite, as witness the great second type of chronic arthritis.

For the treatment of non-union, or pseudarthrosis, many expedients have been recommended. Among these may be mentioned massage, tapping the fragments with a hammer, setons, counter-irritation, weight bearing, rubbing the ends of the bones together, perforating them with a drill, wiring, plating and doweling. The last four expedients are still employed. The bone dowel has many advocates, and doubtless will continue to be employed in special cases, but otherwise the inlay bone graft bids fair to displace other means of treatment in the ordinary case of ununited fracture. It possesses the one great advantage that it is a normal tissue in its physiological place. The dowel is inserted into the marrow canal, where it does not belong, displaces its volume of marrow tissue, and probably must be eventually absorbed.



FIG. 20.—Photograph, of stained slide from sagittal section of knee of dog, resected 922 days previously. Large pieces of bone had been removed. Bony union.

BONE TRANSPLANTATION

The subject of bone transplantation is an interesting one, and has attracted much attention. The discussion,

at first more or less academic, recently with the development of the operative treatment of fresh fractures and of ununited fractures, has attained a very real and practical importance. Difference of opinion as to details still exists, and if one would employ the treatment intelli-

gently, one would do well to possess oneself of a knowledge of the general principles governing its use.

Two things are well settled and we shall mention them before proceeding with the discussion: 1, Live bone from another animal, or from another person, should never be used. It will practically always result in failure; 2, In the presence of suppuration, the transplant will almost always, if not invariably, be cast out.

If a piece of bone be removed, and be immediately

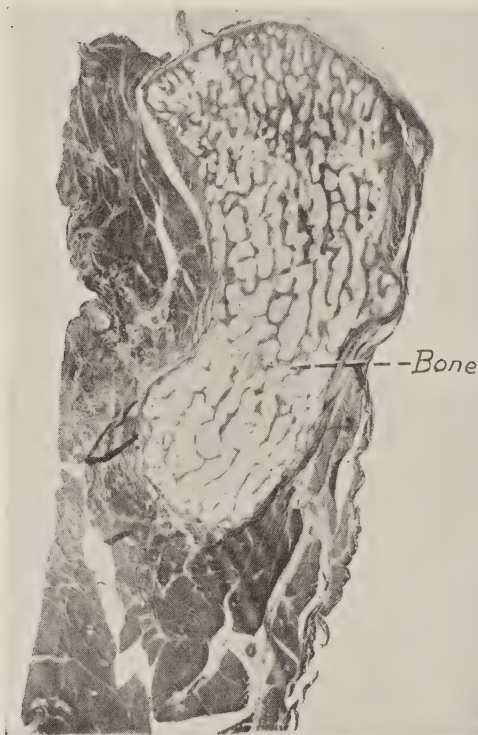


FIG. 21.—Stained slide of section of piece of condyle of femur buried fresh in the thigh muscles of the animal from whom it had just been removed; 922 days after burying. The fragment had not decreased greatly in size. Most of the bone is alive, some of it is dead. The trabeculae are somewhat sparse.

buried again, in the same animal with aseptic precautions the bone dies.²⁵ Whether it dies immediately after removal,

²⁵ COWAN AND ELY: "A Study of Buried Bone," *Journal of Orthopaedic Surgery*, 1919, i, 103.

ELY, LEONARD W.: "Experimental study of Buried Bone," *Annals of Surgery*, 1919, lxx, 747.



FIG. 22.—Stained slide of section of piece of head of tibia buried fresh in the thigh muscles of the animal from whom it was taken, and recovered 473 days later. The fragment had decreased decidedly in size, and is well encapsulated. The bone tissue is small in amount, the trabeculae are scant, the cortex is thin and in places is wanting. Most of the bone is dead, but many of the trabeculae show live bone cells, especially near their margin—'border apposition.' The marrow is about one-half fibrous and one-half fatty.

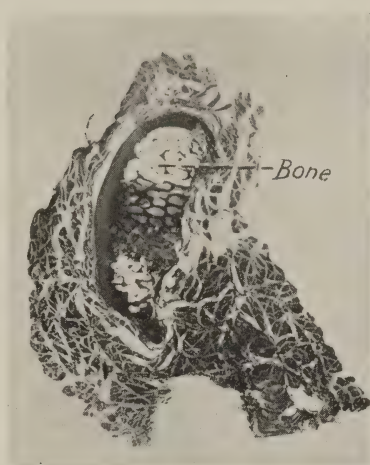


FIG. 23.—Photograph of stained slide of section of a piece of the condyle buried immediately in the thigh muscles of the animal from which it had just been removed, and recovered after 374 days. It had decreased in size. Most of the bone is dead, but many of the trabeculae show life at their edges and there are a few trabeculae under the cartilage, lying in the midst of dead bone, whose basement substance stains sharply in contrast to that of the dead bone, and whose cells also stain.

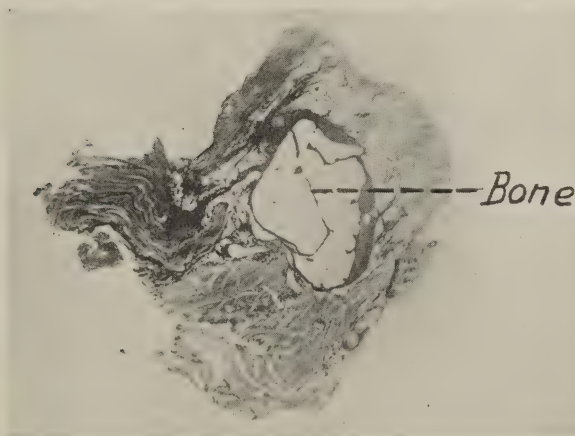


FIG. 24.—Photograph of a stained slide of a cross section of a fragment of the femoral condyle, buried fresh in the thigh muscles of the animal from which it had just been removed, and recovered after 1103 days. The fragment had decreased markedly in size. It was a mere shell and cut easily with the knife. Even the shell had been partially replaced by fibrous tissue. The bone trabeculae are very few and small—mere fragments—but they are all alive.

or whether it lives for a short time is not settled, but that it can live long enough for the blood vessels which are necessary for its nourishment to push their way into it, is so improbable, that we are justified in concluding the death of the graft to be invariable, until positive proof to the contrary shall be adduced. All the experimental evidence

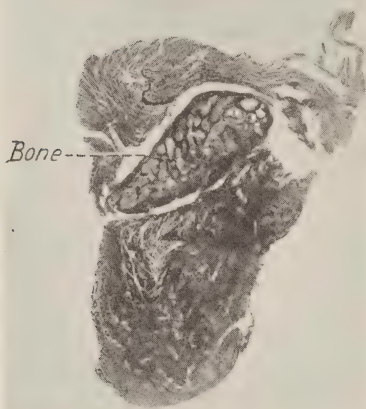


FIG. 25.—Cross section of piece of condyle of femur, boiled before being buried in the thigh muscles of the animal from which it was taken and recovered after 150 days. The fragment had decreased greatly in size and is incapsulated by fibrous tissue. No bone cortex is present except for a short distance. The marrow spaces are bounded by the connective tissue capsule. The trabeculae are about the normal number and size, but most of them are dead. A few however show live bone at their margins.

points in this direction. Those who have examined buried bone are practically unanimous on this point. The evidence on the other side is clinical.

From the moment of imbedding of such a piece of bone two distinct processes arise, one of absorption and one of rebuilding, and, according to circumstances, one of these processes will exceed the other. If the bone have been placed in the soft tissues, where it has no function, although at all times evidences of new bone formation may be seen on the edges of the trabeculae, it will slowly decrease in size and become rarefied, and finally will disappear. It may even last for several years, but its fate is always the same. On the other hand, if the bone be buried in bone, where it will have function, it will persist indefinitely. The same processes are set up in this as in the other, but the process of repair outweighs that of absorption. Even after

years, areas of dead bone are still visible in it.²⁶ Nicety of technique will influence the outcome. The bone must be held firmly in contact with the receiving bone, and immovable, or it will share the fate of the graft in the soft tissues.

If boiled bone be employed for the transplant the same thing occurs except that its absorption will be more rapid, and hence, when employed as a graft between two bones, it is not quite so likely to be replaced and rebuilt as is live bone. Barth²⁷ years ago, basing his opinion on experimental research, declared that, as all transplanted bone died and served only as a scaffold for new bone, the employment of live or of boiled bone was a matter of indifference. Clinical experience caused him to modify his opinion, and it has caused others to modify theirs. Whether a bone grafted in contact with another bone persists, seems to depend upon the rapidity with which it is vascularized.

If a piece of bone with periosteum be examined seventeen days after it has been buried alive, its bone and marrow are seen to be dead. The marrow is without blood vessels. *At the periphery, however, where the fragment is covered by periosteum, blood vessels are seen pushing their way into the bone, and evidences of bone production are seen on the edge of the trabeculae.* This explains the superiority of the living graft to the boiled one; the soft tissues at the surface probably live, are quickly vascularized, and thus hasten the vascularization of the graft.

The above question has a double interest, as bearing on the employment of a graft covered with periosteum. The periosteum acts not only as soft tissue for vasculariza-

²⁶ ELY, LEONARD W.: "Ankylosing operations on the Tuberculous Spine; An Examination of two Specimens in the Laboratory," *Journal of the American Medical Assn.*, 1917, lxviii, 183.

²⁷ BARTH: *Archiv. für klinische Chirurgie*, 1893, xlv, 409; 1894, xlviii, 466.

tion, but possibly also as a matrix for the production of new bone.

Albee, who has done much to popularize the use of the inlay bone graft and to perfect the technique of its insertion, insists that it should be composed of spongy and

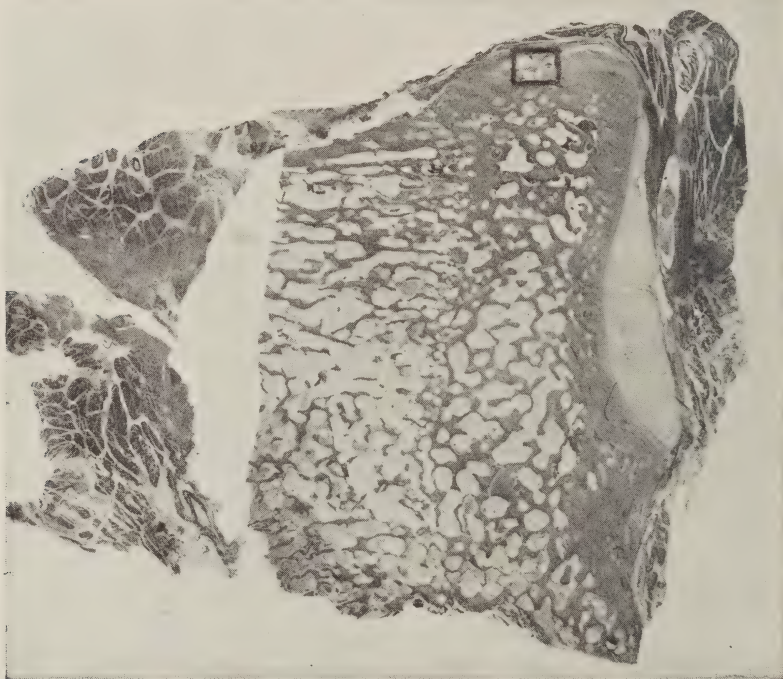


FIG. 26.—Section of condyle of femur of dog, removed at knee-joint resection, and buried immediately in his muscle; removed seventeen days later. Photograph of stained slide, about ten diameters. The bone and marrow are dead, the bone lacunæ are empty. Over the inked square the bone is provided with periosteum. Joint cartilage on the right.

dense bone, and should be covered by periosteum, and that like tissue should be applied to like.

Other rules also should be observed. In old ununited fractures the conditions should be made as much like those of a fresh fracture as possible. If the ends of the two fragments are sclerosed—eburnated—they must be removed,

and the marrow canals beyond must be opened. Healthy bone should be employed for the graft. Its new bed should be prepared before it is removed, and after its removal it should be inserted as quickly as possible, without unnecessary handling, and under strict aseptic precautions. The motor saw should be kept cool to prevent killing the graft



FIG. 27.—This low power photomicrograph, taken of the bone in the inked square in the preceding illustration shows well how all activity in bone formation in the dead buried fragment is carried on through the medium of the imbedding tissues. A blood vessel, B, is pushing its way from the periosteum into the dead bone, and is starting up the formation of new bone. New bone trabeculae, T, T, are forming on the surface of the fragment where it is covered by periosteum, and right close to the surface, where blood can reach them, the bone cells stain.

by heat. A motor saw makes the operation much easier, but the graft may be removed with hammer and chisel. The graft must be fitted accurately in place and must be firmly secured there.

Subsequent immobilization must be furnished by a splint. In old ununited fractures we shall have no assist-

ance from a splint laid down under the periosteum; hence, until enough new bone has been laid down across the gap to stand all ordinary strain, external splints must be provided to prevent refracture. In fresh grafted fractures, splints will be found necessary for a longer time than in simple fractures which have not been operated upon, but in old ununited fractures the time is much longer, running up to

three or four months or more, and depending upon the expected strain. For example, after operation for ununited fracture of the neck of the femur, apparatus should be worn for at least eight or ten months.

A piece of the antero-medial cortex of the tibia, less often its crest, or a piece of split rib, is usually chosen for the graft. Sometimes, especially with fracture of the tibia, a sliding graft is used. A small piece of bone

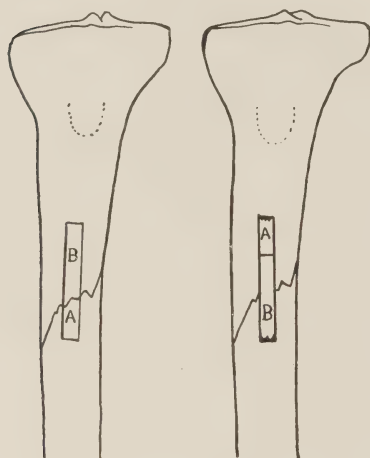


FIG. 28.—This shows how the large fragment is slid across into the gap left by the removal of the small one, and how the small one is used to fill in the gap left proximal to the large one.

is removed on one side of the fracture, and a large piece on the other. The large piece is then simply slid across the gap and the small piece is employed to fill the space left at its far end. This procedure obviates the necessity of two wounds and is quite feasible, provided that the bone tissue of the graft in the vicinity of the fracture is normal in structure.

To avoid the possibility of fracture of the bone from which the graft was taken, this bone also must be splinted for some time after the operation. Removal of the crest

of the tibia weakens the bone much more than does removal of a piece of the anteromedial cortex. A twin saw or a single saw may be employed.

In operating on the upper extremity reliable assistants can hold the limb after the insertion of the graft. In fracture of the femur a special fracture table is almost indispensable, so that the splint may be applied without disturbing the graft. Success depends largely upon attention to details.

With a fresh fracture one carries the incision down to the bone, incises the periosteum longitudinally, strips it back enough to permit approximation of the fragments, and saws out a gutter in the cortex. In old compound fractures the stripping back of the periosteum will occasion more difficulty. It is not necessary to remove all the scar tissue in these cases, but the ends of the bones must be bared, so that the scar tissue no longer lies between them. Both marrow canals must be opened, so that the blood vessels in each can make their way into the graft.

Some operators emphasize the importance of stitching the periosteum of the graft to that of the receiving bone. Others do not find this necessary.

The great advantage of the dowel in fresh fractures is its function as a splint, when it is properly fashioned, but its greatest availability is in old fractures of the femoral neck. In old fractures of the shafts of bones the inlay graft is much to be preferred.

With an old infected compound fracture it is advisable before operating to allow an interval of two or three months after all suppuration shall have ceased. With war wounds the injection of antitetanic serum should precede the operation.

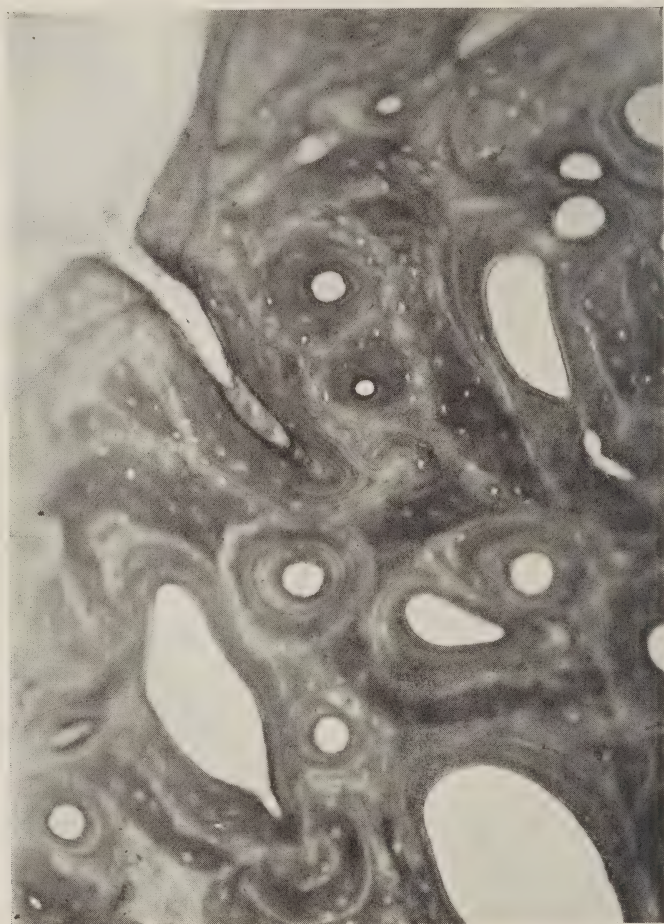


FIG. 29.—Low power photomicrograph of stained slide from a tibial graft used in an Albee spine operation two years before death from intercurrent disease. The graft was united firmly to the vertebral spines. Note the different staining reactions of the live and dead bone, the empty lacunæ, and the blood vessels pushing in from the surrounding soft tissue.

As far as possible the employment of all non-absorbable substances, such as metal nails, screws, or wire, should be avoided.

Other materials have been employed as substitutes for bone in these operations, notably, ivory and the somewhat

cheaper walrus tooth.²⁸ They are not as satisfactory as bone.

Besides its employment in the treatment of fractures, the bone graft has many uses, notably in the treatment of diseased and flail joints to produce ankylosis, and in the

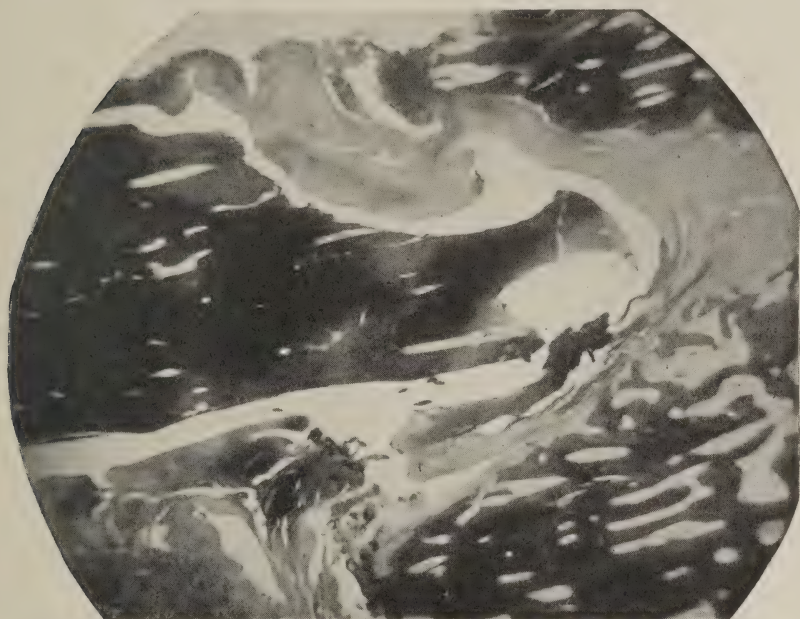


FIG. 30.—Low power photomicrograph of a pseudarthrosis between a bone graft and the bone into which it is doweled. The graft is received into a cup-shaped cavity lined by fibrous tissue. At the surface of the fibrous tissue is a tissue, provided with villi, not to be distinguished from a synovial membrane. The bone of the dowel, as well as that of the receiving cup, is dead.

Albee operation for tuberculosis of the spine. It has been recommended also for a variety of other conditions, and enthusiasts predict its wide availability in the future.

When bone covered with cartilage is buried in the soft tissues, the cartilage persists in good condition long after

²⁸ KOENIG, FRITZ: "Ueber die Implantation von Elfenbein zum Ersatz von Knochen-und Gelenkenden," *Beitraege zur klinischen Chirurgie*, 1913, lxxxv, 19.

the bone has died. It seems to derive sufficient nourishment from the surrounding tissue and does not suffer from being cut off from a direct blood supply. Considerable work has been done experimentally in the transplantation of cartilage, but no practical results ever have been obtained in grafting it where its presence is of real importance, on the joint surface of the end of the bone.

Reports of the transplantation of joints have been made.²⁰ The results of one case were so good for a while that great hopes of the operation were raised, but the hopes were not fulfilled, and joint transplantation has given place to other measures.

ANKYLOSIS

The word ankylosis, or anchylosis, is derived from the Greek *agkulos*, crooked, but the meaning of the term as generally employed, is stiffness or loss of motion in the joint. As a rule we do not employ it to describe the stiffness

²⁰ (a) REHN, EDWARD: "Regeneration des Knochenmarks. . . bei Gelenktransplantation," *von Langenbeck's Archiv.*, 1921, xcvii, Heft 1, 557.

(b) "Epiphysentransplantation." *Münchener medizinische Wochenschrift*, 1911, lviii, 2586.

AXHAUSEN, G.: "Ueber den histologischen Vorgang bei der Transplantation von Gelenkenden." *Archiv. für klinische Chirurgie*, 1921, xcix, 1.

LEXER, "Ueber Gelenktransplantation," *Verhandlungen d. deutsch. Gesell. f. Chir.*, 1910, ii. *Medizinische Klinik*, 1908, 817.

KLAPP, R.: "Ueber Umpflanzung von Gelenkenden," *Archiv. für klinische Chirurgie*, 1911, xcvi, 386.

HELFERICH: "Zur Frage der Transplantation des Intermediärknorpels," *Münchener medizinische Wochenschrift*, 1911, lviii, 2796.

HAAS, S. T.: "The Transplantation of the articular end of bone, etc." *Surgery Gynecology and Obstetrics*, 1916, xxiii, 301. "The Experimental transplantation of the epiphysis, etc." *Journal of the American Medical Association*, 1915, lxxv, 1965.

HELLER, E.: "Versuche ueber die Transplantation der Knorpelfüge." *Archiv. für Klinische Chirurgie*, 1917-18, cix, 1.

present as a physical sign of an active joint inflammation, but only that remaining after the inflammation has run its course. The subject has received various classifications, of which the following is perhaps the best: A true ankylosis is one in which the impairment of mobility is caused by change in the joint tissues themselves; in false ankylosis it is caused by change in the tissues outside the joints. The term contracture is gradually replacing the latter.

Complete ankylosis means an absence of motion, incomplete means a restriction of motion. The former is always bony, though a dense fibrous ankylosis may simulate it clinically. An incomplete ankylosis may be due to fibrous adhesions between the bones, or between one or both of them and the capsule, as in the first great type of chronic arthritis, or it may be due to a change in the shape of the bone ends so that they no longer fit each other, as in the second great type of chronic arthritis.

In order to treat a case of ankylosis intelligently, it is advisable for the surgeon to have a fairly clear idea of the anatomical changes as well as of their cause, and of all the peculiarities of the various joints. No absolute standard of treatment has been established, and every case must be judged on its merits. We do not get much help here from animal experiments. It is almost as hard to produce bony ankylosis in a dog's knee as it is to prevent union of a simple fracture of the shaft of one of his bones.³⁰

The opinion is general that bony ankylosis follows a resection of a joint in man. It often follows resection of the knee, if care be taken with subsequent immobilization. I think the hip is rather hard to ankylose. Bony ankylosis is not to be expected after resection of the ankle or the

³⁰ ELY, LEONARD W.: "Experimental Resection of the Dog's Knee Joint," *Annals of Surgery*, 1919.

shoulder, and it is notoriously hard to secure in resection of the elbow. Exact information is lacking in the toe joints,



FIG. 31.—Bony ankylosis of wrist following an old closed infectious arthritis of unknown etiology.

but the end of the metatarsal may be cut off in cases of hallux valgus with little fear of ankylosis, though some stiffness may remain.

It is easier to stiffen the joint of the adult than that of the child. Normal joints become stiff after immobilization. The exact anatomical cause of this stiffness is hard to determine. Some say that fibrous adhesions form in the joint, and hasten to remove splints in fracture cases and to carry out early active and passive motion. Others maintain that a normal joint will never become permanently ankylosed, no matter how long it may be immobilized, and that the restriction of motion is caused by contraction of the tissues outside the joint. It is hard to say where the truth lies. Neither side seems to have any actual proof. In former times I held the latter view very strongly, but I am not quite so sure as I was.

As to the treatment of ankylosis, competent authorities agree that no attempt should be made to mobilize a joint which is the seat of an active inflammation. Motion is the worst possible thing for such a joint, and we should assist nature in her efforts to provide rest. Pain is nature's great conservative symptom. If a joint is painful, its owner will try to keep it at rest. When all evidences of active inflammation are absent, the case is different.

Two things are important to know in a case of ankylosis. The first is its type, whether it is complete or incomplete, and whether caused by adhesions between the ends of the bones, or by their distortion. This is determined by an

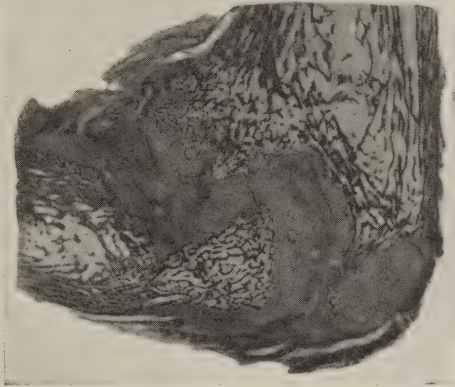


FIG. 32.—Old fibrocartilaginous ankylosis of the elbow-joint. One can easily realize the effect of any treatment other than operative upon a joint like this.

examination of the joint, and by the Röntgen picture. The second thing is the probable cause of the ankylosis. This is determined by a careful history, and by the examination of the patient himself as well as of his joint.

Ankylosis from distortion of the bone ends is usually caused by the second great type of chronic arthritis, or by old joint fractures. Fibrous ankylosis results from tuberculous, gonococcic, syphilitic, staphylococcic, streptococcic arthritis, and from other forms of arthritis also. Streptococci and staphylococci may also cause a bony ankylosis. Syphilis probably never does. Simple intraarticular fractures may result in bony ankylosis probably through the building of a bony bridge in the capsule. A rare form of bony ankylosis has been described in the elbow, following a slight injury, without any evidence of fracture. It seems to have been essentially an ossification of the capsule. I have seen it follow an unreduced dislocation of the hip in an elderly man.

According to Ollier³¹ and Mauclaire,³² after a bony ankylosis, the structure of the bone at the site of the former joint changes. The diameter slowly lessens, the spongy bone is absorbed, the cortical bone thickens, the proximal and distal medullary canals extend until sometimes they join, the lymphoid marrow gives place to fatty marrow, and finally a typical section of a shaft of a long bone may be established. I have never had the opportunity to examine a specimen in the laboratory, but I have watched with the Röntgen rays the changes in the architecture for as long as ten years after knee joint resections,

³¹ OLLIER: "Dict." *encycl. des sciences Medicales*, Paris, 1870, Ankylose, p. 191.

³² MAUCLAIRE: "Nouveau Traité de Chirurgie," Paris, 1909. Ankyloses, p. 235,

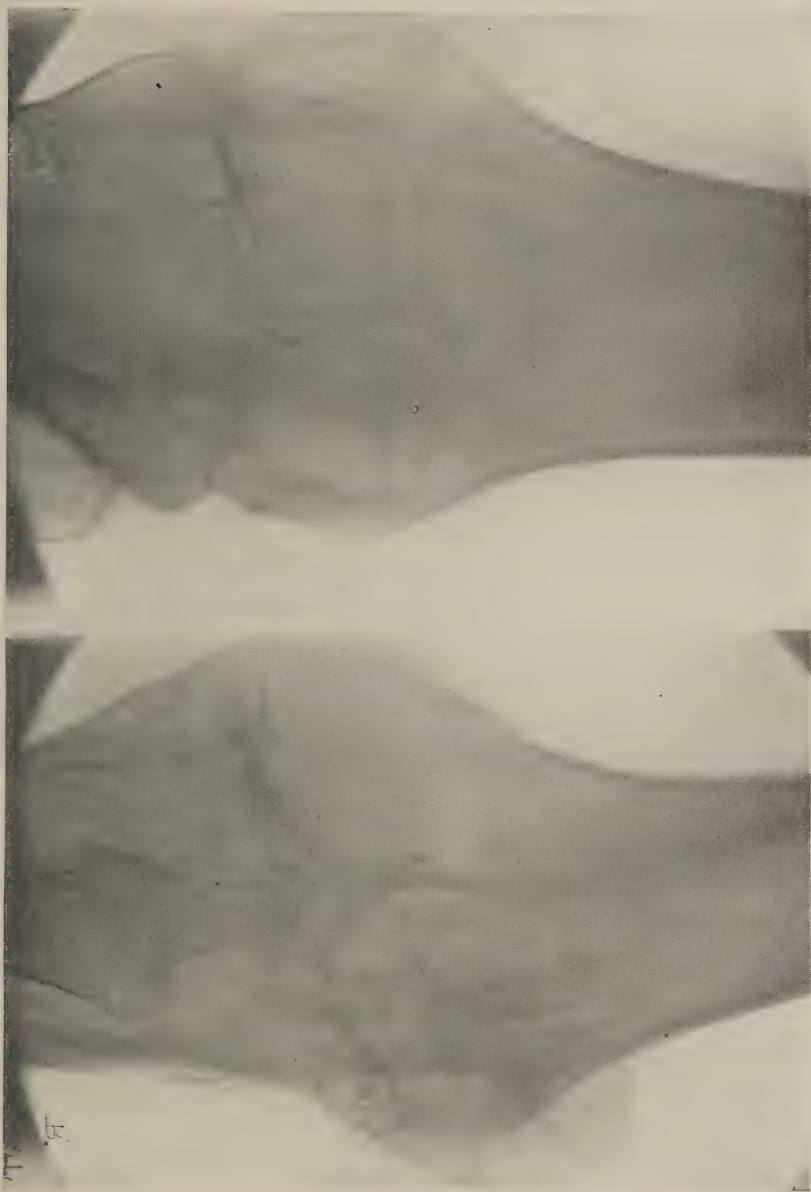


FIG. 33.—Tuberculous knee ten years after resection.

and have seen the bone remodelled along these lines, but apparently not completely canalized. Complete canalization takes a long time. Spongy bone has been observed



FIG. 34.—Ankylosis caused by new bone in the joint capsule. Motion was restored in this case by the removal of the bone, and the insertion of a fascial flap over its base.

two and a half years after the resection of a tuberculous knee.³³

THE TREATMENT OF ANKYLOSIS naturally falls under the head of that of the diseases which cause it, but certain aspects of it may be considered here.

³³ WAGSTAFF, MR.: *Path. Soc. Trans.*, 1868-69, xx, 264:

If there be reason to believe that tuberculosis was the cause, most authorities advise that the ankylosis be left absolutely alone. Even many years after all active symptoms have subsided, foci of infectious material may remain locked up in the bone marrow, or in the fibrous adhesions of the joint, capable, if they be set free, of lighting up the disease afresh.

The ankylosis following joint fracture may sometimes be improved by remodelling the bone ends by operation. Each case must be judged on its merits. Sometimes what appears to be a firm ankylosis turns out to be a plate of bone in the capsule continuous with one of the articulating bones. If it be removed, and a flap of fat or of fascia be swung over its base, free motion may be restored. In dealing with the knee joint it is well to find out if the tibio-femoral articulation, or the femoro-patellar, or both joints together, are involved.

When the ankylosis is caused by fibrous adhesions, these may be broken up forcibly under an anæsthetic (*brisement forcé*), and then the joint may be immobilized in a different attitude. It may be necessary to repeat the manœuvre several times. In point of fact not much can be expected of this method.

Physical therapy—baking, massage, hydrotherapy, gentle active and passive motion, etc.—at present has quite a vogue. Fibrolysin is more or less of a medical relic.³⁴

ARTHROPLASTY

The modern attempts at forming a new joint are usually dated from Ollier. Helferich followed Ollier. In this country the operation was first strongly advocated by Murphy. The subject has been investigated and de-

³⁴ SIDORENKO, P.: "Experimentelle und klinische Untersuchungen ueber die Wirkung des Fibrolysins etc." *Deut. Z. f. Chir.*, 1911, cx, 89:

veloped by Payr, Sumita, Allison and Brooks, Baer and many others.

The simplest form of the operation of arthroplasty is the division of ankylosing tissue, possibly supplemented by a shaping of the bones. Little is to be expected from this. Its poor results gave rise to attempts to supplement it with the interposition of various substances, organic and inorganic. Ollier turned in periosteum between the two bones. Others interposed muscle flaps, fat and fascia flaps, animal membranes of various kinds, and even inorganic substances. The flap of fat and fascia is the one now most employed.

Most writers affirm that temporomaxillary arthroplasty gives the best results. Perhaps this is because the operator on this articulation has but one object, to mobilize it. In other joints the fear of instability enters into the calculation. An unstable knee, for instance, is much worse than a stiff one, though many knees have been mobilized without losing their stability. The hip offers a better prognosis than the knee.

THE OPERATION.—Artificial ischæmia is not advisable, though Murphy employed it. The joint is opened so as to provide free access to it. In the case of a fibrous ankylosis, all the fibrous adhesions are dissected away, and also the contracted and cicatricial capsule. This is important. In a bony ankylosis the bone is divided at the site of the former joint, and the bone ends are trimmed to a shape as near their former shape as possible. Plenty of the bone must be removed. In old fibrous ankylosis also, the bones should usually be reshaped. All bleeding must be checked. The tissue to be interposed is then sutured carefully over one articulating surface. If an attached flap is used, it is partially dissected from its bed in the neighborhood, and

swung between the bones to be sutured. The wound is closed.

The period of the subsequent immobilization is a question of circumstances, and largely of opinion. It is possible to err on either side. Obviously motion should begin as soon as practicable, but not soon enough to disturb the healing of the wound. Payr begins motion in about one week, using traction after the first forty-eight hours. Baer puts his cases up in plaster of Paris for about four weeks. I incline to an average of ten days to two weeks. Active as well as passive motion should be carried out. Electrical stimulation and massage of the atrophic muscles may begin almost immediately. Small hæmatomata should be aspirated. The patient's coöperation is almost a *sine qua non*, and patience and interest on the part of the operator also.

In knee operations long medial and lateral incisions are the favorite. A separate long incision from near the proximal end of the lateral incision lays bare the fascia almost up to the trochanter. A strip of this about 10 cm. wide and 20 cm. long is then incised on its proximal, anterior and posterior margins, dissected clear of the vastus lateralis with care, and still attached at its distal border, is drawn through a tunnel made between this incision and the first lateral one. It is sutured carefully over the entire end of the femur, so that it lies between the femur and the tibia, and between the femur and the patella as well. Before the wound is closed all bleeding must be stilled.

Corner's long anterior incision³⁵ with splitting of the

³⁵ CORNER, E. M.: "The Surgery of the Knee." *Journal of the American Medical Association*, 1914, lxiii, 1069.

patella, should be admirably adapted to this operation. (Fig. 96.) Kirschner's³⁶ has also been recommended.

In many cases of knee ankylosis, the tibio-femoral joint is normal and the entire trouble resides between the femur and the patella. The flap then would be interposed solely between these two.

In hip joint operations Baer favors the anterior incision, running distally from the anterior superior spine, about 15 cm. long between the tensor fasciae latae and the sartorius. Sprengel's incision³⁷ or Smith-Peterson's slight modification of it,³⁸ which add to the anterior incision one running from the anterior superior spine along the ilium just distal to the crest, gives a better exposure. (Fig. 91.) Murphy favors a large lateral U-shaped incision five inches wide with its base proximal and its convexity distal. (Fig. 92.) The trochanter occupies the centre of the flap. The flap includes the skin, fat and fascia lata and is reflected proximally. A threaded curved needle, passed around the trochanter draws a Gigli saw after it, and the trochanter is then divided and drawn proximally. This gives an excellent exposure of the joint. A flap of fat and fascia is dissected from the large original flap.

In the elbow, medial and lateral incisions may be employed or a long posterior one with transverse sawing of the olecranon and its subsequent wiring or suturing.

In the shoulder, the ordinary anterior incision will be found useful, between the deltoid and pectoralis major, or the posterior incision of Kocher.³⁹

³⁶ KIRSCHNER: "Ein neues Operationsverfahren zur schonenden Eroeffnung des Kniegelenks." *Beitr. z. klin. Chir.*, 1910-1911, lxxi, 703.

³⁷ SPRENGEL: "Zur operativen Nachbehandlung alter Hüftresektionen," *Archiv. f. klin. Chir.*, 1898, lvii, 837:

³⁸ SMITH-PETERSON, M. N.: "A new supra-articular, sub-periosteal approach to the hip-joint." *American Journal of Orthopædic Surgery*, 1917, xv, 592.

³⁹ KOCHER, THEODOR: "Mittheilungen aus der chirurgischen Klinik in Bern." *Arch. f. klin. Chir.*, 1888, xxxvii, 777.

In temporomaxillary arthroplasty, Murphy⁴⁰ advocated an L-shaped incision, the perpendicular arm about 4 cm. long running immediately in front of the ear, from the zygoma, and the horizontal about 2 cm. long, along the upper border of the zygoma. He removed about 1 cm. from the head and neck of the bone, and employed a fascial flap from the temporal muscle.

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⁴⁰ MURPHY, JOHN B.: *Journal of the American Medical Association*. 1914, lxii, 1785.

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SECTION II.

ACUTE OSTEOMYELITIS AND ARTHRITIS

CHAPTER I

ACUTE SUPPURATIVE HÆMATOGENOUS OSTEOMYELITIS

OF the four usual causes of inflammation, mechanical, thermal, chemical, and bacterial, the last two only are of great importance in inflammations of the bone marrow. Its situation protects it from ordinary degrees of heat and cold, and also from all injuries except fracture.¹ After a simple fracture the changes in the marrow are essentially those incidental to the repair of the bone.

Injury seems to do little permanent damage to the marrow. If the marrow be curetted from the shaft of a laboratory animal it quickly regenerates, and, after a comparatively short time shows no trace of the injury.² It is important to remember this fact, on account of the persistent tendency to ascribe to trauma a leading rôle in most diseases, acute and chronic, of bone as well as of other tissues. As our knowledge becomes more exact, trauma usually sinks more and more into the background.

As to the influence of chemical agents, the evidence for one, such as phosphorus, is positive, that for others, such as toxines, is largely presumptive.

The cause of osteomyelitis in an overwhelming proportion of cases is infection, and the chief bacterial infectious agents are the staphylococcus and streptococcus pyogenes, the tubercle bacillus, the bacterium coli commune, the gonococcus, the treponema pallidum, the diplococcus, the typhoid bacillus, and the pneumococcus.

¹ Extreme low temperature may, of course, cause necrosis of the marrow.

² ELY, LEONARD W.: "Regeneration of the bone marrow. Bone and Joint Studies I." Stanford University 1916, Published by the University.

It is customary to divide cases of osteomyelitis into acute and chronic, and this division, while by no means exact, is convenient and will be adopted here. Some cases are acute from the start, run a typical course, and end in recovery or in death. Others are essentially chronic. Still others start with all the earmarks of an acute disease, and then settle down into a chronic stage, to last indefinitely, or possibly to recover under appropriate treatment. Again, what has been from the beginning a chronic disease may at any time take on all the appearance of an acute one.

ACUTE OSTEOMYELITIS

The one great cause of acute osteomyelitis is infection. The infection may be carried in from the outside, as with compound fractures; it may make its way into the bone from neighboring tissues, as with acute arthritis or deep suppurations; or it may be blood borne. The last constitutes a special disease of itself, with a well defined clinical picture, and will be considered first.

What is commonly known as osteomyelitis, or acute osteomyelitis, is a suppurative inflammation of the bone marrow, occurring almost always in children or in adolescents, with an acute onset, febrile course, and marked constitutional reaction, and ending in the death of a greater or less amount of marrow and of bone. Its cause is usually the staphylococcus pyogenes aureus, less often the staphylococcus pyogenes albus or the streptococcus pyogenes, and rarely the bacterium coli commune. The organism may exist alone, or the infection may be a mixed one. With invasion by streptococci a marked tendency to involvement of the neighboring joint has been noted. In many instances the organism can be cultivated from the blood.

Occasionally the offending organism is brought from

some known suppurating focus somewhere else in the body, usually its place of origin is not known. Its favorite port of entry has been assumed to be an abrasion of the skin, however small, and the fact that the staphylococcus albus is the most common infecting agent lends color to this assumption. The tonsil has been under suspicion, not only on account of the occasional sequence of the disease on scarlet fever, but also because of the causal relation of the tonsil to acute and to chronic arthritis.

The importance of trauma as a contributing cause has been much debated. The results of animal experiments are contradictory. Clinical opinion can be found on both sides of the subject. At present we must consider the question as not proved.

The sequence of the disease upon an infectious disease, especially upon scarlet fever, has been noted, and also its sequence upon chilling of the surface.

As has been said, acute osteomyelitis is essentially a disease of childhood and adolescence. It is most frequent in the second decade of life, and many cases occur in the first and third decades. Lexer taught that its location in the bones was dependent upon the arrangement of the blood-vessels about the epiphysial line. Klemm³ considers acute osteomyelitis as simply a suppurative inflammation of lymphoid tissue, similar to that of lymphoid tissue anywhere else in the body, and only modified and aggravated by the rigid bony shell in which it runs its course. I advanced this reason for the occurrence of marrow tuberculosis ten years ago.⁴

Acute osteomyelitis almost invariably begins in the

³ KLEMM, PAUL: "Die Osteomyelitis des Kindesalters," Berlin, Verlag von S. Karger, 1914.

⁴ ELY, LEONARD W.: "Joint Tuberculosis," William Wood and Company, 1911.

metaphysis⁵ of one of the long bones, especially in that of the femur and of the tibia, less often in that of the humerus. The purulent inflammation spreads in the spongy bone, killing the marrow and the bone as it goes, and the process then adopts one of four different courses.

1. THE TYPICAL SEVERE SPREADING OSTEOMYELITIS. —The inflammation travels widely in the spongy bone in the general direction of the central marrow canal, gains this, and involves its marrow for a greater or smaller distance, sometimes reaching the epiphysial line at the other end of the bone. It also passes through the minute canals in the cortex, and comes to the under surface of the periosteum. It runs along between the cortex and the periosteum, dissecting them apart. If an operation has not been undertaken by this time, or death has not intervened, the pus breaks through the periosteum, burrows in every direction, and finally makes its way to the surface. We have then the necrotic shaft—the sequestrum—filled with pus, and lying within the periosteum, bathed in pus, and connected with the surface by sinuses.

Death, from sepsis, from ulcerative endocarditis, or from metastases in the kidneys or in other organs, would probably have closed the scene; if not, then with the free discharge of pus, the disease settles down to the chronic stage. In areas the periosteum may become adherent to the necrotic bone beneath it. After a few weeks, bone formation begins in or under the periosteum, and proceeds until the necrotic shaft is enclosed in a more or less complete shell of bone (the involucrum) perforated by holes for the discharge of pus—cloacæ, sewers.

A stage is thus reached which, without outside aid, will last indefinitely. The dead shaft, locked up in its rigid

⁵ The metaphysis is that portion of the spongy bone which lies shaftward from the epiphysial line.

case, is incapable of absorption, and remains permanently as a foreign body. In the end the patient may die from the effects of the prolonged suppuration.

Not always does the disease reach the extreme limits detailed above. It may be halted at any point by nature's protective reaction in the marrow, usually by a fibrosis. The wall of defence may slowly be reinforced by bone, and this, with the periosteal bone, forms a perforated box, in which lies the cylinder of sequestered shaft, more or less continuous with it in structure.

2. THE SUPERFICIAL FORM.—In this the disease, starting as in the preceding form, near the epiphysial disc, and probably near the cortex, never reaches the central marrow canal, but breaking through the thin cortex near the end of the bone, gains the under surface of the periosteum, and spreads shaftwards beneath it. It kills the superficial part of the cortex, and filling the space between it and the periosteum with pus, lifts the periosteum off, perforates it, and then, burrowing through the soft tissues, makes its way to the surface. This form of the disease is wont to be less severe than the preceding.

3. THE CIRCUMSCRIBED FORM.—Brodie's abscess.⁶ In this form the disease remains localized in the metaphysis. An abscess forms, usually not of great diameter, and becomes walled off by dense bone. It may remain indefinitely stationary, or, according to some writers, may break out later, and spread through the bone, like the other forms. During its existence it may give rise at any time to a sterile effusion in the joint, as may the other three forms. It is said to be one of the causes of the so-called intermittent synovitis, or hydroarthrosis. Presumably Brodie's abscess is caused by bacteria of a low degree of virulence.

⁶ See an interesting article by WALTER M. BRICKNER: "Chronic medullary abscess in the long bones," *Annals of Surg.*, 1917, lxx, 483.

4. **THE ARTICULAR FORM.**—Starting at or near the same place as the three preceding, the disease shows a marked tendency to spread towards the joint, perhaps spreading shaftward also. It may perforate the epiphysal cartilage, and involve the epiphysis, and then, having killed the joint cartilage, in whole or in part, break into the joint and become an arthritis. This is the “acute epiphysitis” of some writers. The epiphysis may be separated by the inflammation beneath it. The disease may break directly into the joint without perforating the epiphysal cartilage. In either case, the result is the same. According to most writers, this type of case is usually caused by the streptococcus, less often by the pneumococcus or the typhoid bacillus.

It is seen therefore that all four forms of the disease are the same in their pathological characteristics. They differ only in their details. Their course may be checked, modified, or changed by operative interference.

OSTEOMYELITIS (PERIOSTITIS) ALBUMINOSA (SEROSEA) is a rather rare form of the disease, usually occurring in the femur, with an acute and severe onset, and then a milder and chronic course which may last for several months. Suppuration does not result, but sequestra form, and when at length the fluctuating swelling is opened, the fluid is found to be thin, serous, synovia-like. The disease is caused by the same organisms as the other forms.

SYMPTOMATOLOGY.—Acute osteomyelitis usually begins suddenly with the customary symptoms of an acute infection, chill, high fever, rapid pulse, etc. Sometimes the onset is preceded for a few days by headache, malaise, and pain in the limbs. The patient complains of severe pain in the vicinity of a joint, most often the knee, but not in the joint itself. Motion of the joint is usually not very pain-

ful, but pain is caused by prolonged pressure over the affected region, or by percussion. Redness and swelling, often œdematous, soon appear, and the œdema may continue distal to the seat of trouble. As soon as the process breaks through the cortex, a fluctuating swelling appears. The neighboring joint sometimes fills with fluid, which in the majority of cases is sterile. The leucocyte count is very high, and cultures of the offending organisms may often be made from the blood. The subcutaneous veins are prominent, the adjacent lymph nodes are enlarged.

The patient rapidly becomes worse, and often sinks into the so-called typhoid state, with coated tongue, delirium, etc. The fever as a rule is continuous. A fluctuating temperature is a sign of metastases. The urine is concentrated, and often contains albumin. Cutaneous eruptions may be present.

Such a picture is comparatively easy to recognize. On the other hand, swelling may be absent, as may all local physical signs of disease, and the delirium and coma may be so early and so profound that the patient is not in a position to complain of pain. If the disease breaks into the joint, the evidences of an acute arthritis are added.

THE DIAGNOSIS in most cases is easy. The acute onset, with high fever, usually with a chill, the painful swelling in the neighborhood of a joint, extremely sensitive to pressure, with dilated superficial veins, and local œdema, present a characteristic picture. When the disease is in the region of the hip, buried deeply in the muscles, its recognition is not so simple. The appearance of the fluctuating swelling removes all doubt.

The X-rays give us, as a rule, little help during the first week. After that, changes in the bone are evident. These show in the increased permeability of the bone to the rays,



FIG. 35.—Acute, suppurative, hæmatogenous osteomyelitis of the femur in the chronic stage.

more or less irregular, of course. In the later stage the new bone in the periosteum can be seen on the plate, and the sequestra surrounded by rarefied areas.

The effusion into the neighboring joint, occasionally occurring in the early stages of an acute osteomyelitis, the

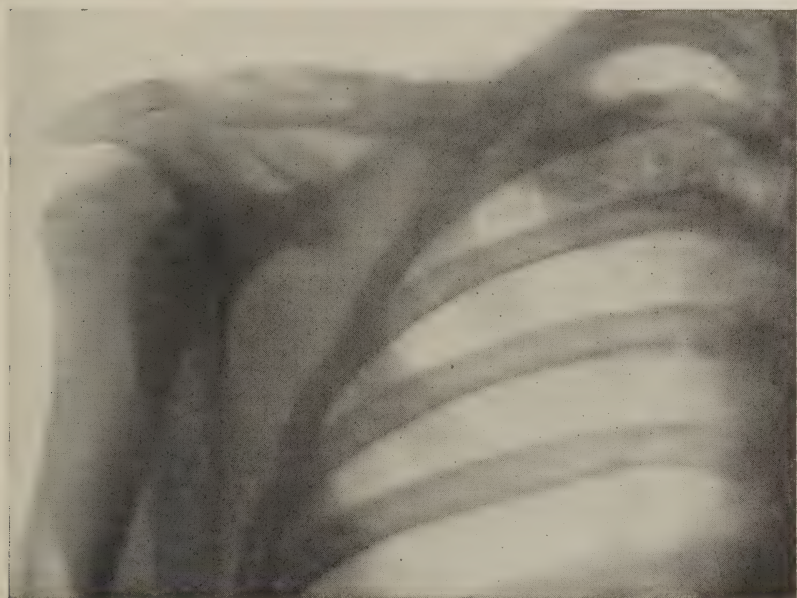


FIG. 36.—Acute, suppurative, hæmatogenous osteomyelitis of the clavicle, in the chronic, stage.

so-called sympathetic synovitis, is usually sterile. Aspiration will determine this.

An acute suppurative arthritis shows a swelling in the joint itself, and possibly for a short distance on each side of it, rather than on one side alone. Motion is much more painful. Fluid in the joint is an early phenomenon of acute arthritis, and can be found by aspiration. When pus is found, operation reveals its source in either instance.

Acute inflammatory rheumatism is differentiated by its fleeting nature; by involvement of several joints in

succession, the first clearing up as the next is attacked; by the acid sweats; and by its reaction to salicylates.

The severe cases, with few evidences of local disturbance, but with marked constitutional involvement, early delirium and rapidly deepening coma, offer the greatest difficulty. Sometimes only the necropsy clears up the origin of what has been diagnosed as a pyæmia of unknown origin.

Typhoid fever has a characteristic onset, with a delirium usually not appearing early, intestinal symptoms, a Widal reaction, and an absence of leucocytosis. The typhoid state is rather late in typhoid. To occasion any confusion in osteomyelitis it would need to appear early.

THE PROGNOSIS AS TO LIFE, as well as to the time of complete recovery, depends largely upon the promptness of treatment and its thoroughness. With early operation the majority of patients recover. Some patients die quickly from toxæmia, even before the formation of pus. Others die after a short time from sepsis, ulcerative endocarditis, metastases in the kidneys, etc.; others after a longer time from the effects of the prolonged suppuration, amyloid degeneration, or metastases in other bones. The persistence of bacteræmia after thorough drainage is a bad sign, but not necessarily a fatal one.

THE TREATMENT is invariably operative, at the earliest possible moment, as soon as the diagnosis can be made, and without waiting for fluctuation.

Under complete narcosis the long incision is made deliberately in the muscle planes down to the periosteum, and this is slit open. If pus be found it must be followed up as far as it has traveled. With involvement of the marrow beneath, the cortex usually loses its glistening appearance and shows dead white, while pus, instead of blood, oozes

from the minute apertures of the Haversian canals. Later, the cortex turns a dirty brown or blackish.

In no circumstances must fear of infecting the marrow induce one to stop here. A long strip of the cortex should be removed, the marrow canal should be inspected, and if it be found infected, the removal of the lid of bone over it should be continued until the limits of the disease have been reached. It is not enough to open simply the central marrow canal. The metaphysis, where the process started, must be opened as well. In the early stages the marrow may be found only intensely congested, perhaps with yellow streaks running through it; later it will consist of yellow or greenish pus. If we have reason to suspect pocketing of the pus under the periosteum on the side of the bone opposite to that opened, counter openings should be made.

Authorities differ as to the next step. Some operators stop here, either leaving the wound open or stitching the skin to the periosteum, to ensure its staying open. Some curette the marrow. Some not only curette, but also pack the cavity. Some immediately resect all the diseased bone. Most operators postpone the bone removal. The Carrel-Dakin treatment seems particularly appropriate after operation.

Nichols advises that the bone be not removed until new bone formation in the periosteum, the involucrum, has well begun. This time can be determined by thrusting a needle into the periosteum, or more accurately by examining a piece of it under the microscope. The new bone imparts a crackling sensation, with a slight resistance. In the forearm and in the leg, where the diseased bone has a splint in the other bone, Nichols sets the time as about two months after the first operation; in the femur and in the

humerus, where no such splint is present, the time should be somewhat longer.

Other operators wait longer before removing the sequestrum, but they all agree that too long an interval must not be allowed to elapse. If we wait too long, the bone in the involucrum becomes very dense. The reparative processes in dense bone are very poor, and in cases that have been postponed too long, the cavity left by the removal of the sequestrum may remain indefinitely.

If early sequestrotomy be practiced, a splint should be applied, on account of the danger of fracture of the involucrum, a fracture that sometimes will not unite. If time for the formation of a solid involucrum be permitted to elapse, the removal of its steep sides at operation will hasten healing.

When it comes to removing the sequestrum, the dead bone is not as a rule found perfectly separated, but united at its ends to the live bone and here and there adherent to the periosteum. These attachments must be divided. Sometimes, also, a considerable amount of bone must be chiseled from the involucrum, to permit of the removal of the sequestrum.

When the disease has involved only the superficial part of the cortex, the problem is much simpler. In such a case the sequestrum may be in the form of a long sliver of bone which can be removed easily.

After the removal of the sequestrum the disease settles down into its chronic stage, and it is customary to allude to this stage as chronic osteomyelitis. It really is the stage of repair of the acute disease, a stage of repair which is prolonged by mechanical conditions incident to the presence of the bony shell which prevents healing, as well as to the presence of pieces of dead bone unavoidably left behind.

In favorable cases during the healing of the wound small sequestra are thrown out. After healing has taken place, to the accompaniment of slight pain and increase of temperature, pus pockets may form in and near the scar, and, rupturing, heal again after discharging small sequestra. This may go on indefinitely, but the ordinary period may be said to be a year.

The most troublesome cases are those in which, after a reasonable time, the processes of repair come to an end, leaving a cavity in the bone, with sclerosed walls, which will not fill in. Sometimes if the steep walls of this cavity be removed, giving it a shallow cup shape, repair will start up again, and will be completed. In other cases this procedure fails, especially when the cavity is near a joint, where its obliteration is difficult. The problem is then a difficult one, and has been attacked in various ways.

Schede⁷ recommended filling the cavity with an aseptic blood clot, followed by almost complete closure of the wound and the application of a protective tissue between it and the voluminous dressing. It is difficult or impossible to prevent infection of the blood clot, and in the hands of others than its originator, this treatment has not been successful. Hamilton's⁸ sponge graft seems to have no advocates nowadays, nor Senn's⁹ decalcified bone chips.

Good results have been obtained with Neuber's¹⁰ oper-

⁷ SCHEDE: "Ueber die Heilung unter dem feuchten Blutschorf." *Dtsch. med. Wochens.*, 1886, xii, 389.

⁸ HAMILTON, D. J.: "On sponge grafting." *Edin. Med. Jour.*, 1881, xxvii, 385.

⁹ SENN, NICHOLAS: "Healing of aseptic bone cavities." *Am. Jour. of Med. Sci.*, 1899, xcvi, 219.

¹⁰ NEUBER: "Zur Behandlung starrwandigen Höhlenwunden." *Arch. f. klin. Chir.*, 1896, li, 683.

ation. Neuber cleaned out the bone cavity, trimmed off its steep walls as much as possible, and turned in flaps of skin and soft tissue, securing them to the bone by nails, straps and invagination sutures.

Perhaps the most promising procedure is, after trimming the walls of the cavity and rendering them, as far as possible, aseptic, to fill them with some special form of paste. The exact chemical combination of the paste is probably not a matter of great importance. As von Mosetig-Moorhoff¹¹ says, the "plombe" is merely a temporary substitute. Von Mosetig-Moorhoff, after thorough disinfection and drying of the walls of the cavity, filled it with a paste composed of a sterile mixture of iodoform 40 parts, and oil of sesame and spermaceti 30 parts each. He then closed the wound.¹²

The treatment of the superficial form of the disease is usually much simpler than that of the preceding. As a rule only the superficial portion of the bone is killed, and this only in a limited area. Healing follows slowly after the removal of the dead bone.

THE CIRCUMSCRIBED FORM OF OSTEOMYELITIS—BRODIE'S ABSCESS.—The abscess should be cleaned out thoroughly, and its sclerosed walls should be chiseled away. The employment of some paste may be advisable to promote the healing in of the cavity, *e.g.*, Mosetig-Moorhoff's, Beck's or "bipp." The formulæ of Beck's two pastes are: No. 1, bismuth subnitrate (arsenic free) 33 per cent., vaselin 67 per cent. No. 2, bismuth subnitrate 30 per cent., vaselin 60 per cent., paraffin (120° melting point) 5 per cent., white wax 5 per cent. The formula for

¹¹ VON MOSETIG-MOORHOFF, DR. R.: "The elimination of cavities in operative wounds." *Surgery, Gynecology and Obstetrics*, 1906, iii, 547.

¹² See also CLOPTON, M. B.: "The diagnosis and treatment of osteomyelitis." *Surgery, Gynecology and Obstetrics*, 1915, xx, 6.

bipp is: Iodoform 16 ounces, bismuth subnitrate 8 ounces, liquid paraffin 8 fluid ounces, or a sufficient quantity. If Beck's paste or bipp be used, one must be on the lookout for symptoms of bismuth poisoning.¹³ The possibility of iodoform poisoning with bipp and Mosetig-Moorhoff's pastes must not be forgotten. Perhaps vaselin and paraffin, without iodoform and bismuth, would be as efficacious as with them.

In operations on old sterile bone abscesses, the wound may be closed immediately.

¹³ BECK, EMIL G.: "Bismuth paste in chronic suppuration." St. Louis C. V. Mosby Co., 1915, 182.

DREESMANN: "Ueber Wismuthvergiftung." *Münch. med. Wchnschr.*, 1901, xlviii, 238.

ELY, LEONARD W.: "A fatal case of bismuth paste poisoning." *Med. Record*, 1912, lxxxii, 119.

FREILICH, ELLIS B.: "Bismuth poisoning following bismuth paste injection." *J. A. M. A.*, 1917, lxviii, 111.

MAYER, LEO, AND BAEHR, GEORGE: "Bismuth poisoning." *Surg., Gynec. and Obst.*, 1912, xv, 309.

MORISON, RUTHERFORD: "The treatment of infected suppurating war wounds." *Brit. Jour. Surg.*, 1916-17, lv, 660.

MUHLIG, F.: "Ueber Wismuthvergiftung." *Münch. Med. Wchnschr.*, 1901, lxviii, 592.

SIGMUND, ERDHEIM: "Ueber Wismutintoxikation bei Behandlung nach der Methode von Beck." *Wien. klin. Wchnschr.*, 1912, xxv, 749.

VACCAREZZA, RAUL F.: "Las intoxicaciones por el subnitrate de bismuto." *La Semana Med.*, 1919, xxvi, 366.

CHAPTER II

ACUTE SUPPURATIVE HÆMATOGENOUS ARTHRITIS

THE ARTICULAR FORM.—It is quite likely that the infectious material may be carried to the synovial membrane in the first place, and thus that a suppurative



FIG. 37.—Photomicrograph of bone and cartilage from a section of acute suppurative arthritis, following operation on a case of suppurative inflammation on the shaft of the tibia. Degenerating cartilage on the right, bone marrow on the left. Observe how the inflammatory process in the marrow is eating its way into the cartilage, and how it has already consumed most of the bone. Only a few small trabeculae are left.

arthritis may precede the osteomyelitis, or may exist alone without involvement of the bone. The result in either case is much the same, though if the synovial membrane alone be affected, the treatment may be different.

In the treatment of these acute suppurative arthritides the problem is not, as in an uncomplicated osteomyelitis, a simple one of thorough drainage, but we are led to take risks by the hope of a movable joint. Again, in the case of

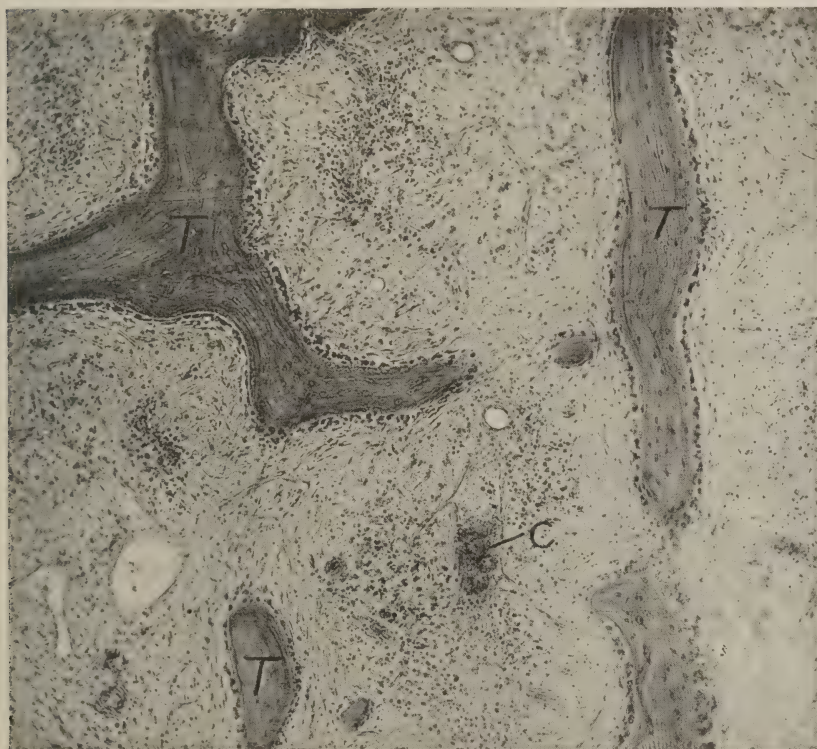


FIG. 38.—Suppurative inflammation in the bone marrow in the region of the joint. T, T, T trabeculae; C, colony of pus cocci in a small vessel. We know that in this case the bone is rapidly being destroyed, but the margins of the trabeculae present an appearance typical of what is usually described as productive osteitis.

a child, we must not do any more injury than is absolutely necessary to the epiphysial cartilage.

Thorough drainage is demanded here as in the other forms of the disease, but it is well not to be too radical about gouging out the bone, especially about the epiphy-

sial cartilage. If constitutional symptoms be not severe, and if aspiration of the joint have revealed only a few leucocytes or even a few cocci, we may postpone opening it, possibly following Murphy's advice,¹ washing it out and injecting its cavity with a 2 per cent. solution of formaldehyde in glycerin. The injection may perhaps be repeated once or twice if the constitutional symptoms and the local physical signs justify it.

If the joint be full of pus, containing many cocci, little is to be gained by temporizing. It should be opened and thoroughly drained. Any drains which we insert must be left in, of course, as long as necessary, and yet we must remember that joints which have had tubes in them for any length of time usually become ankylosed. On the other hand, it is not likely that a joint, invaded by an osteomyelitis in the vicinity serious enough to damage the cartilage badly, will ever possess enough function to justify much risk for its preservation. Cartilage scraped off from limited areas of the articular surface of animals' bones sometimes reforms, but nothing that I have ever seen in my specimens makes me think that it is replaced after it has been destroyed by disease. We come back, then, to the principle that an acute hæmatogenous suppurative arthritis requires free drainage.

In disease of the knee or hip, traction may promote the comfort of the patient, and possibly may help in the treatment. Later on some such splint as the Thomas may be of service when the lower extremity is affected.

It is sometimes impossible in an acute hæmatogenous suppurative arthritis to decide whether the synovial membrane alone is involved or whether the infection springs

¹ MURPHY, JOHN B.: "Metastatic gonorrheal arthritis of the knee." *Surgical Clinics of John B. Murphy*, 1912, i, 825.

from a marrow focus in the immediate vicinity. If the bone involvement is so minute as to escape detection, it may be ignored.

A SUPPURATIVE HÆMATOGENOUS ARTHRITIS is occasionally caused by the diplococcus of pneumonia. The disease comes on during a pneumonia or in early convalescence, and is often fatal.² It may arise in the course of a general infection, and very rarely it is primary. Its treatment is the same as that of the preceding, but cures by aspiration have been reported. If the constitutional symptoms warrant it, this should be tried before more radical measures.

A HÆMATOGENOUS ARTHRITIS occasionally is observed as a sequel of certain acute infectious diseases, notably scarlet fever, measles, smallpox and influenza.³ The inflammation may consist of a mild synovitis, single or

² BEZANCON, F. AND GRIFFON, V.: "Arthritis expérimentales a pneumocoques par infection générale et sans traumatisme articulaire." *Soc. d. Biol. Comp. Rend.*, 1899, li, 709.

CAVE, EDWARD J.: "Pneumococcic arthritis." *Lancet*, 1901, i, 82.

DUNN, L. A. ROBINSON, H. BETHAM AND FLETCHER, H. MORLEY: "Five cases of purulent pneumococcic arthritis in children." *Lancet*, 1903, ii, 316.

ELY, LEONARD W.: "A case of pneumococcus infection of the hip." *Med. News*, 1905, lxxxvi, 930.

HERRICK, JAMES B.: "Pneumococcic arthritis." *Am. Jour. Med. Sci.*, 1902, cxxiv, 12.

HORSFALL, C. E., CAMPBELL: "Pneumococcus arthritis; vaccine treatment; recovery." *Lancet*, 1918, ii, 556.

PASTEUR, W. AND COURTAULD, L.: "Primary pneumococcal arthritis." *Lancet*, 1906, i, 1747.

RAW, NATHAN: "Pneumococcus arthritis with notes of seven cases." *Brit. Med. Jour.*, 1901, i, 1803.

SECRETAN, W. BERNARD, AND WRANGHAM, WILLIAM: "Pneumococcic arthritis." *Brit. Med. Jour.*, 1906, i, 915.

³ FRITSCHS "Ueber Gelenkerkrankungen bie Scharlach und Masern." *Beit. z. klin. Chir.* 1911, lxxii, 101.

SCHUELLER, MAX: "Ueber Bacterien bei metastatischen Gelenkentzündungen." *Archiv. f. klin. Chir.*, 1884-5, xxxi, 276.

multiple, which soon clears up entirely, or it may be more severe, with a plastic exudate. This latter form is frequently seen after influenza, and may result in almost complete ankylosis, from the dense adhesions in the joint. The arthritis may be suppurative, and not to be distinguished from the acute streptococcic form already described. Its treatment is the same.

CHAPTER III

SUPPURATIVE OSTEOMYELITIS FOLLOWING COMPOUND FRACTURE

THE suppurative osteomyelitis which follows infection from a compound fracture differs in its course and in its clinical manifestations from the hæmatogenous form, but the fundamentals of the pathological process of the two are much the same. The infection, of course, is always a mixed one, and, starting from any part of the bone, it spreads in every direction. In former days it was frequently fatal, but since the introduction of the modern methods of treatment of compound fractures, except in war wounds it is rarely seen. Otherwise its manifestations are often mild in their nature.

THE TREATMENT in the first place is prophylactic. A compound fracture which is considered clean may be treated expectantly, but at the first sign of infection, it should be thoroughly opened up. All foreign material and necrotic or loose bone fragments should be cleaned out, but bone fragments still attached to the periosteum should be left. Their presence is said to promote the union of the fracture. Provision should be made for drainage, and possibly some form of wound disinfection should be installed—Carrel-Dakin. If in spite of our efforts, the infection spread, amputation may be necessary.

Non-union frequently follows infected compound fractures. It is well not to undertake any operation for this until after the wound has been healed for several months. Otherwise we run the risk of lighting up the infection afresh.

CHAPTER IV

TYPHOID OSTEOMYELITIS AND ARTHRITIS

TYPHOID OSTEOMYELITIS.—It appears from the investigations of Fraenkel¹ and others that the bone marrow is a favorite domicile of the typhoid bacillus, after clinical recovery as well as during the actual course of the disease. Sometimes it causes an osteomyelitis which is wont to affect the superficial portion of the cortex, and may or may not result in suppuration. The infection may be a simple one or it may be mixed. The shafts of the long bones, especially of the ribs or of the tibia seem to be affected by preference, but the disease is by no means rare in the spine. It is commonly called a periostitis when it occurs in the shafts of the long bones.

Typhoid osteomyelitis usually develops in the third or fourth week of the disease, or at any time after the typhoid has run its course, and its onset is manifest ordinarily by a rise of temperature and a painful swelling of the bone. If suppuration results, the abscess should be thoroughly cleaned out and drained. The milder cases of a pure typhoid infection often disappear spontaneously, and need no other treatment than rest and protection from injury.

TYPHOID ARTHRITIS

An arthritis is an occasional complication of typhoid fever, coming on in the late stages of the disease, or during convalescence. The fluid in the joint may be sterile, or it may contain typhoid bacilli, alone or mixed with pus cocci.²

¹ FRAENKEL, EUG.: "Ueber Knochenmark and Infektionskrankheiten." *Muenchener medicinische Wochenschrift*, 1902, xlix, 561.

² ELLIS, A. G.: "As Experimental Study of Joint Affections Induced by the Typhoid Bacillus," *Jour. of Infect. Dis.*, 1909, vi, 181.

Marrow involvement is probably, as a rule, slight. The chief pathological changes appear to be in the synovial membrane.

Typhoid arthritis most often occurs in the hip, and usually is not very painful. Dislocation of the hip is a rather frequent result.

THE TREATMENT consists in rest and protection. In order to avoid a dislocation of the hip, the attitude of extreme flexion and adduction is to be avoided. If the joint be much distended with fluid it should be aspirated. With secondary infection by pus germs an operation becomes necessary, though, as the infection is probably synovial alone, it may possibly be checked by washing out the joint cavity with normal salt solution, and then injecting it with a mild antiseptic such as a solution of formaldehyde.

TYPHOID SPINE.—This is an acute affection of the spine, coming on in the late stages of a typhoid, or during convalescence, and generally in vigorous young men. Its pathology has not been definitely established, for no case ever has come to necropsy, though in Rugh's case a necropsy was done nine years after the attack. Osler taught for many years that it was a neurosis, but the clinical picture, the Röntgen plate, and the occurrence of a kyphosis in one or two cases indicate that it is essentially an osteomyelitis, perhaps with an arthritis as well. No case ever has gone on to suppuration, and we assume that the offending organism is the typhoid bacillus.

The disease may be mild and insignificant, and may pass as a "lumbago" practically unnoticed; it may be very severe. Its onset may be sudden or slow. Usually, in the late stages of a typhoid or during convalescence, the patient

complains of stiffness in his back and of pain in the lumbar region. The pain in the severe cases is horrible, and is increased by the slightest motion. The patient is unable to turn in bed and cries out if the bed is only slightly jarred.

The pain often comes in paroxysms, preceded perhaps by a rise in temperature. It may remain localized in the back, or it may run around the abdomen, or into the extremities. It may shift.

With the symptoms of an actual bony lesion may go those of a neurosis, or even of a marked hysteria—paræsthasias, muscular contractions, changes in the reflexes, etc.

After a longer or shorter interval the symptoms slowly subside, and the patient recovers completely within a year. The milder cases run a short course.

THE TREATMENT may be summed up in one word—rest. The severe cases demand recumbency, possibly with the immobilization reinforced by a gas-pipe frame, covered with canvas, to which the patient is strapped. Morphine, or even a whiff of chloroform or of ether may be necessary to quiet the paroxysms of pain.

For the milder cases, and for the later stages of the severe ones, sufficient immobilization is furnished by a light steel spinal brace, or by a plaster jacket. The white-hot thermo-cautery, flicked quickly over the lumbar region, relieves the late pain and stiffness, and its application is relished by the patient.

CHAPTER V

GONOCOCCIC OSTEOMYELITIS AND ARTHRITIS

THE gonococcus can cause an inflammation of the bone marrow, or of the synovial membrane, or of both. Practically all authorities speak of a gonorrheal periostitis. I believe that this is not correct. The inflammation is in the superficial portion of the bone marrow and not in the periosteum.

On account of the difficulty of recovering and growing the gonococcus, our observations on the disease are largely clinical. The corollary to this is that much difference of opinion prevails in regard to the disease. The cause of gonorrheal arthritis is of course the gonococcus, but the diagnosis is made far more often without the demonstration of the organism than with it. The disease, as would be expected, seems to be more common in men than in women.

It is customary to speak of acute and of chronic gonococcic arthritis and to teach that while its incidence is usually in the florid stage of the urethral infection, at about the time of invasion of the deep urethra, it may nevertheless come on at any time during the course of an old chronic infection in the deep urethra. I think that this is an error; that a gonococcal arthritis is always an acute arthritis, and that it almost invariably, if not invariably, comes on during the acute stage of the urethral infection. I have never seen the gonococcus recovered from a case of chronic arthritis, and believe that in the old cases of chronic arthritis caused by a lesion in the deep urethra, the streptococcus is the offending organism. Baer,¹ however found the gonococcus

¹ BAER, W. S.: "Painful heels." *Johns Hopkins Hospital Bulletin*, 1905, xvi, 264.

in a number of old spurs under the calcaneus. In acute arthritis the organism has a fleeting life; one day aspiration of the joint, especially immediately after an acute exacerbation, detects it; the next day it may be gone. One might take either side of a wager on its presence or absence.

On account of a paucity of pathological material we know little of the finer changes in the bone and joint. In the region of the joint the bone usually shows rarefaction under the X-ray. Farther from the joint, immediately under the periosteum, bone absorption and bone production go hand in hand. This often results in irregular spurs and exostoses.

The synovial membrane is inflamed, succulent, congested and villous. The exudate in the joint, while occasionally purulent or possibly serous, usually is fibrinous or plastic. It results sometimes in dense fibrous adhesions, which limit or abolish joint motion.

The typical and characteristic invasion of the disease, not by any means invariable, however, is as follows: For a day or two several joints swell up and become slightly painful, one clearing up as the next is involved. Then, suddenly, the disease attacks one or perhaps two joints and stays there. The swelling and pain are great, with an increase of local temperature and marked sensitiveness. The joint is distended with fluid. *Edema of the tissues in the neighborhood may be said to be almost characteristic.* Constitutional symptoms as a rule are not marked. High fever with pronounced constitutional symptoms, usually spells mixed infection.

The large joints are more frequently involved than the small ones. Everyone has his own ideas as to the relative frequency of involvement of the various joints. Personally I look for it in the man most often in the knee or ankle,

in the woman most often in the wrist, but then I have not seen many cases in which I was sure of the diagnosis. Sterno-clavicular involvement is most suggestive of the gonococcus.

The disease has no definite course. Recovery may take place after a longer or shorter time, either complete or with much or little remaining stiffness. Endocarditis, iritis and "gonococcic septicæmia" accompany the arthritis quite frequently, possibly because they all are an evidence of a severe infection, or of a high degree of vulnerability on the part of the patient.

THE DIAGNOSIS as a rule is not difficult. The presence of an acute urethral infection is strong presumptive evidence. The patient rarely attempts to hide this, as he does a chronic one. Acute inflammatory rheumatism has a fleeting nature, acid sweats, and usually a ready reaction to the salicylates. In acute suppurative hæmatogenous osteomyelitis the start of the process is outside the joint. From acute suppurative hæmatogenous arthritis the disease may be differentiated by aspiration. The fluid from a gonococcic joint is sterile or contains gonococci.

TREATMENT.—Some authorities recommend immobilization, others maintain that immobilized gonococcic joints are more likely to become ankylosed. Hot applications are grateful to some patients, cold applications to others. Good results have followed washing out the joint with normal salt solution, or with a solution of protargol or argyrol.

In aspirating or washing out a joint it is well to incise the skin and superficial fascia with a scalpel, and then use a large calibre needle or a trocar.

Specific antigonococcic sera and vaccines have been employed often during the past ten years, and have been

highly praised. Apparently they are of great value. Recent investigations, however, indicate that the specificity is of no importance. The result of the use of non-specific proteins is said to equal that of specific proteins.

Whatever local treatment be instituted, the prime indication in these cases is the treatment of the primary focus



FIG. 39.—The so-called gonorrheal periostitis of the tubercle of the calcaneus.

in the genito-urinary tract. It is a strange thing that the urethral symptoms often lessen as the joint becomes involved, to reappear as the joint condition improves.

A peculiar condition sometimes is seen in old cases of deep urethral infections. The patient complains of pain in the soles of his feet, where the plantar fascia is inserted into the tubercle of the calcaneus. Standing and walking are painful. Pressure by the thumb brings out the sensitiveness very prettily. The gait is characteristic. The

patient walks as if he were treading on eggs. The Röntgen rays may or may not show a roughening of the calcaneal tubercle. Baer recovered the gonococcus from the tubercle in one or two cases.

Attention to the lesion in the deep urethra usually causes a prompt disappearance of the symptoms. Vesiculotomy is often necessary. Massage of the prostate may be tried. Possibly strapping, or a sole plate sharply arched up under the anterior part of the calcaneus, may also be of benefit.

Some surgeons advise an operation. The incision may be made on the medial aspect of the heel. It is carried down to the bone, and the suspected tissue is scraped out. Operation is rarely necessary if the deep urethra be cleared up.

A similar condition of painful heels, not so marked nor so characteristic, is occasionally observed in patients with a focus of infection in their teeth or tonsils. Patients with flat feet also may complain of painful heels, but they have not the "egg-walking" gait. A spur is occasionally seen in the Röntgen picture of normal calcanea. It gives no symptoms, and requires no treatment.

CHAPTER VI

ACUTE INFLAMMATORY RHEUMATISM

STRICTLY speaking, this is not a joint disease, but a general disease characterized by painful joint swellings and a marked pyrexia. Its cause never has been demonstrated. Numerous investigators^{1,2,3,4,5,6,7,8,9}, have isolated organisms from the blood and from the joints, but have not succeeded in proving their causal relationship. Some believe that the disease is a toxæmia or a mild form of pyæmia. A modern view is that it is essentially an anaphylactic reaction, similar to that, for instance, against diphtheria antitoxin, but more severe. A strong array of evidence points to the tonsil as the seat of the infection, whatever it may be.

Acute inflammatory rheumatism is characterised by a

¹ ACHALME, P.: "Sur un signe de diagnostic précoce des attaques et des rechutes de rhumatisme articulaire aigu." *Arch. Gén. de Méd.*, 1902, exc, 257.

² BEATON, R. M., AND WALKER, E. W. A.: "The Etiology of acute rheumatism and allied conditions." *Brit. Med. Jour.*, 1903, i, 237.

³ BEATTIE, JAMES M.: "A contribution to the bacteriology of rheumatic fever." *Brit. Med. Jour.*, 1906, ii, 1781.

⁴ BEATTIE, JAMES M.: "The Micrococcus rheumaticus. . ." *Brit. Med. Jour.*, 1904, ii, 1511.

⁵ LEWIS, MORRIS J., AND LONCOPE, WARFIELD T.: "Experimental Arthritis and endocarditis..." *Assn. Am. Phys. Trans.*, 1904, xix, 457.

⁶ POYNTON, FREDERICK J., AND PAINE, ALEXANDER: "The etiology of rheumatic fever." *Lancet*, 1900, ii, 861, 932.

⁷ POYNTON, F. J., AND PAINE, ALEXANDER: "Some further investigations and observations upon the pathology of rheumatic fever." *Lancet*, 1910, i, 1524.

⁸ ROSENOW, E. C.: "The Etiology of acute rheumatism, articular and muscular." *Jour. Infec. Dis.*, 1914, xiv, 61.

⁹ TRIBOULET AND COYON: "Recherches bactériologiques concernant un cas de rhumatisme fébrile mortel. . ." *Soc. de Biol. Comp. Rend.*, 1897, xlix, 1000.

high, irregular, remittent temperature, a high colored acid urine, and sweats. The larger joints as a rule, suffer most. They swell, one after another, and are exquisitely sensitive to motion and to touch. The swelling is circumarticular as well as articular. The adjacent tendon sheaths may also be involved. The joint is red and hot. The great peculiarity of the disease is its fleeting nature. It flits from joint to joint, one joint clearing up as the next becomes involved. Another marked feature is its reaction to the salicylates.

Acute inflammatory rheumatism is rarely overlooked when it is present. Its features are so characteristic that they can hardly be ignored. The diagnosis is employed too often rather than too seldom. Many lives have been lost by failing to recognise an acute suppurative arthritis, and treating it as "rheumatism." It is a good rule never to diagnose anything as rheumatism, until every thing else has been ruled out.

THE TREATMENT belongs in the realm of internal medicine. Splinting may alleviate the pain. There is a history of frequent sore throat, and the tonsils are diseased. These organs should be removed. Not infrequently the disease lights up after tonsillectomy, probably on account of the entrance of organisms through the raw surface.

CHAPTER VII

HYDRARTHROSIS INTERMITTENS

Intermittent Synovitis, Intermittent Joint Hydrops

THIS rare and somewhat mysterious lesion was described by Perrin.¹ It consists of a periodic effusion into one or more joints, usually into the knee. The fluid remains for a few days and then disappears.

The cause is unknown. It has been regarded as a syphilitic manifestation, as a neurosis, and as a reaction of the synovial membrane to a focus of infection in the end of the bone, or in some other part of the body. Coincident disease of the thyroid gland has been noted, and vasomotor disturbances elsewhere. Some observers consider it as a passive effusion, others as a synovitis. It occurs with about equal frequency in men and women.

The joint fluid is usually serous, and contains flakes of fibrin. The synovial membrane shows its customary reaction to the presence of fluid in the joint cavity, and becomes thickened and villous.

The swelling usually comes on rather suddenly, increases for a few days, and then subsides, to return after an interval which may vary in length. Pain, if present, is not usually severe. Fever, dizziness, headache, pain in the limbs, vasomotor disturbances and other constitutional symptoms have been observed. In women a certain relation to the menses has been noted. If several joints are attacked, they may be attacked coincidently or one after another. Occasionally the affection leaves one joint and attacks an-

¹ PERRIN: *Journal de Médecine*. Par. 1845, iii, 82.

other, like acute inflammatory rheumatism. Sometimes the patient can foretell the exact date of his next attack, but again cases which have been regular in their recurrence may become irregular, and vice versa. In women the attacks sometimes have subsided during pregnancy.

TREATMENT.—Immobilization, apparently, is of no avail. As to internal medication the best results seem to have been obtained with quinine and arsenic, although no malarial basis ever has been found for the disease. On the chance that it may be caused by a focus of infection in the bone end, or possibly may be an “anaphylaxis” to a distant focus, a search for a focus should be instituted, and if one be found, it should be removed. The only patient with this affection I ever saw, had evidences of infection in both teeth and tonsils, whose eradication she would not permit.

CHAPTER VIII

TRAUMATIC ARTHRITIS

Traumatic Synovitis

A SIMPLE traumatic arthritis may be caused by an injury to the joint capsule, or by an intraarticular fracture. In either case blood is poured out into the joint cavity, and probably, by its mere presence, irritates the synovial membrane. The membrane becomes thickened, infiltrated, succulent and villous, encroaching somewhat on the cartilage at its border. When the effusion has been absorbed, and the capsule or bone has healed, the synovial membrane returns to normal. A bloody or fibrinous exudate often leaves adhesions behind, with some stiffness which persists for a while.

THE DIAGNOSIS is usually quite simple. An acute arthritis immediately following an injury is practically always a traumatic arthritis. It is not always easy to differentiate between the injury to the capsule—a sprain—and one to the bone—a fracture. Excluding the Röntgen rays, perhaps the two most valuable helps are the ecchymosis and the point tenderness on the bone in fracture. False point of motion and crepitus are hard to elicit unless the fracture is a severe one, and then there will be little difficulty in the diagnosis.

THE TREATMENT of the arthritis complicating fracture is that of the fracture itself, except that if the amount of fluid in the joint be excessive, and cause great pain and possibly difficulty in the reduction of the fragments, it may be aspirated under strict asepsis.

The continued pain and stiffness which follow the healing of a fracture with misplaced fragments in a healthy person, are probably due to the constant spraining of the damaged joint apparatus, viewed as a machine. It is doubtful if any permanent changes are set up in the bone thereby. If, for instance, from an old stiff wrist, following a carpal fracture, the offending fragments be removed, good function often returns. On the other hand, marked stiffness and disability frequently follow a well reduced fracture, especially in an elderly person. In such case a careful examination of the patient, and especially of his other joints will probably reveal evidences of preëxisting bone changes. It is likely that the fracture simply converted a walled-in chronic infectious osteomyelitis into a chronic arthritis, which will be improved by prompt attention to the teeth.

A SPRAIN is the tearing of a ligament, but the term includes also the traumatic arthritis which results, and in the knee this is so dominant that we are wont to allude to the injury as a synovitis, whereas in the ankle we never should think of calling it anything but a sprain.

The cardinal symptoms are pain and restriction of motion, especially of motion in one direction. The joint is swollen and its contour is changed. It contains fluid, which can easily be demonstrated in the knee and in the elbow, with more difficulty in the hip, ankle and spine. In the knee, the fluid floats the patella off the condyles; in the elbow fluctuation is to be sought posteriorly at the sides of the triceps tendon. The pain is made worse by motion which puts the damaged ligament on the stretch. Usually the joint is held in semiflexion.

THE DIAGNOSIS with the help of the Röntgen rays usually is simple. Without it, it is often impossible. The

main thing to establish is that the joint was normal at the time of the injury, in order that one may exclude a disease that was simply aggravated by it.

TREATMENT.—The first indication is to restrict as much as possible the formation of the exudate in the joint, especially in a joint that has a capacious cavity like the knee. This is done by rest, pressure, strapping, bandaging on a splint, etc. Ice bags, or hot compresses also are useful. These measures may be continued for a few hours. Later the important indication is rest, not necessarily rest for the whole joint, but rest for its damaged part. In the later stages, to overcome stiffness, physical therapy—baking, hydrotherapy, massage—may occasionally be advisable.

THE KNEE is usually sprained in its medial ligament, and especially when it is in flexion. Far less often is a crucial ligament torn. Therefore the chief indication, after the acute symptoms have subsided, is to hold the joint in extension, in which position it is rarely injured. This is accomplished by diagonal criss-cross strapping with overlapping strips of adhesive plaster about ten inches in length and an inch or two in width, quite firmly applied over the front and sides of the knee, reaching from a point below the joint to one above the top of the quadriceps pouch. This strapping not only prevents flexion but also exerts pressure. The patient is encouraged to go about, preferably with a cane. His comfort is a good indication of the efficacy of the treatment. If he suffers pain the treatment is not sufficient. Then immobilization probably will be necessary, and subsequent physical therapy.

If a semilunar cartilage be torn, the semidetached piece becomes pinched between the tibia and femur, and the joint is sprained. It fills with fluid, and an attack of “synovitis”

results. Often the knee "locks" in semiflexion, and only can be released by manipulation by the patient himself or by a bystander. Sometimes the deformity is fixed, and cannot be overcome. When the knee has been straightened, it usually swells for a while and is painful. The symptoms slowly subside, and the joint returns to normal. No further trouble may be experienced, but as a rule the accident recurs, possibly many times, and each time it is followed by the symptoms of joint irritation, usually not so severe as those following the first attack. The cause is a twist of the knee when it is in semiflexion. It is a frequent accident among athletes, especially among football players. The medial meniscus is much the more frequently torn.

THE DIAGNOSIS as a rule is not difficult. The Röntgen picture is negative. The patient gives the history of the attacks of locking. If sensitiveness of the anterior portion of the medial meniscus be present in addition, the diagnosis is fairly certain, though surgeons who have often opened the knee agree that absolute certainty is not possible.

TREATMENT.—Various manipulations by the patient are successful in reducing the deformity—shaking the leg on the thigh, while the patient is erect or lying on his face, swimming motions carried out on the floor, etc. The surgeon may aid by shaking the flexed leg with the patient prone, or by flexing, extending and rotating. Jones¹ recommends "acute flexion, lateral deviation, and rotation inwards and full extension." Sometimes it is hard to tell whether the cartilage has been reduced or not. The patient may be able to give us the required information, but there is a peculiar feel that may clinch the matter. If, with the patient supine, the knee be passively flexed and extended, attempted full extension comes to a sort of springy stop as

¹ JONES, ROBERT: "Notes on derangement of the knee." *Annals of Surg.*, 1909, i, 969.

long as the cartilage is out of place, whereas with a normal knee, the stop is sudden and definitive. If all attempts at reduction fail, it is sometimes wise to send the patient home for 24 hours, to see what he can do by himself. If he fail, operation is practically imperative.

When the cartilage is first torn and has been reduced, the knee should be splinted, at least partially, and should be treated with the idea of healing the torn cartilage. There is a reasonable chance of success by these means. With each succeeding slip the chances grow smaller. Most authorities agree that recovery is not to be expected after the cartilage has been out two or three times, but cases have been observed (I have had a personal experience with one) in which recovery has ensued after numerous attacks. This is rare, and there is always an element of risk in the accident. There are times when a simple fall in the street from locking of the joint might be fatal. Most surgeons do not operate after the first slip, especially as the diagnosis is not a matter of certainty. The oftener the accident recurs after this, the stronger is the indication for operation.

JONES'S OPERATION, with the knee flexed, has attained a wide popularity. Strict asepsis is of the first importance. Not even a gloved finger should touch the wound. All sponges should be on holders. Artificial ischæmia is to be employed. The patient's legs hang over the end of the operating table, and the operator sits facing them.

The incision is a curved one. It starts from a point just proximal to the distal border of the patella, and about a centimetre medial to its medial border, runs distally to a point about a centimetre distal to the tibial margin, and then curves sharply medialwards for about two centimetres. It is about eight centimetres long. The incision through the capsule is about half as long. The edges of the wound

are retracted, and the torn piece of cartilage is cut off with a knife, and without traction on it. Jones never ties vessels, always uses a tourniquet, and never drains. The wound is sutured in layers. The joint is immobilized for eight or ten days, and then passive motion and massage are begun.

THE ANKLE is usually sprained by a twist of the foot inward, tearing the external lateral ligament. An excellent treatment, perhaps after twenty-four hours of hot or cold applications, is that with the Cottrell (sometimes erroneously called the Gibney) strapping.

TREATMENT.—The foot is held in dorsal flexion and marked eversion. The plaster, in strips one inch wide, and in two lengths, eight and twelve inches, is to be ready at hand. The first strap (eight inch) starts just behind the metatarsophalangeal joint of the little toe, runs around behind the heel, and ends in the region of the medial malleolus. The second (twelve inch), starts about the middle of the lateral surface of the calf, passes down the leg, under the heel near its posterior part, and ends near the medial malleolus. The third overlaps the proximal half of the first, the fourth the anterior half of the second, and so on until about eight have been applied. A few criss-cross straps reinforce these where they cross at the ankle, and a snug bandage holds them all in place. The immediate relief afforded by this dressing is often remarkable, when it is properly applied.

SPRAIN OF THE ELBOW is rare and demands rest. The same may be said of the shoulder. Subacromial bursitis, with its painful rotation and abduction, is to be suspected in painful injuries about the shoulder.

SPRAINS OF THE WRIST also are probably quite rare, and should be diagnosed only when other lesions have been ruled

out. In this connection three things especially must be borne in mind: 1st, Fractures of the radius or of the carpal bones. The Röntgen rays will detect these. 2nd, Ganglion. This is a rather small, tense, fluctuating swelling found usually on the dorsal aspect of the wrist, slightly to the radial side, or less often on the volar aspect in the immediate vicinity of the radial artery. 3rd, The so-called tenosynovitis or tenovaginitis crepitans, a slightly painful, diffuse swelling, over the posterior and lateral aspect of the distal end of the radius. When the patient actively extends and abducts his thumb, a fine crepitation like the rubbing together of two pieces of silk, is communicated to the examining finger. Circular strapping with adhesive tape, about two inches wide is a good treatment for a sprained wrist.

SPRAINS OF THE SPINE, especially of the spine already affected with a chronic arthritis, are quite common, though on account of the inaccessibility of the spinal joints, the diagnosis is never more than presumptive. Following a severe wrench, twist, or sudden strain, the patient experiences a sudden pain, usually in his lumbar region ("lumbago") or down his thigh ("sciatica"). The pain often is quite severe, and as a rule is accompanied by marked stiffness of the spine. Sometimes the patient is unable to bend forward at all, at other times forward bending is carried out with a lateral deviation. When the sacroiliac joint is involved, perhaps also when the lower lumbar spine is sprained, a Kernig sign is present. An Ely sign² occasionally may be elicited in lumbar arthritis or sprain.

²In certain irritative lesions of the lower lumbar spine, if the patient lie prone, and his knee be flexed, his pelvis will rise from the table. This is the Ely sign, and it is probably caused by the pull forward on the rigid lumbar spine by the rectus femoris muscle.

TREATMENT.—These cases ordinarily are treated with “antirheumatic” remedies as “myositis,” “fibrositis,” “lumbago” or “sciatica.” A Röntgen plate usually will show the evidences of a chronic arthritis, and the main efforts at treatment should be directed at this. (See the chapter on chronic arthritis.) In sacroiliac sprain the X-rays often show a subluxation of the pubic symphysis. In the simple cases, snug strapping about the sacroiliac joints, *below the anterior superior spines* may be all that is necessary. Physical therapy also has its advocates. Sometimes the subluxated pubic joint must be reduced under an anæsthetic. Superextension of the thigh on the affected side will usually be found the best manipulation.

CHAPTER IX

SUPPURATIVE ARTHRITIS FROM WOUNDS

THE joint may be involved directly by the penetration of dirty foreign bodies, or it may be involved secondarily by the spread of an infection from a compound fracture opening into the joint. In the first case the marrow may become infected secondarily from the joint. The infection is of course always a mixed one. At its start it possesses peculiarities depending upon its place of origin, and the nature of the trauma, but once under way, its course is much the same as that of a hæmatogenous infection. The soft parts may become gangrenous at an early stage.

Our ideas on the treatment of joint wounds have been greatly modified during the late war, and, while unanimity has by no means been reached, a fairly well-recognized standard of procedure has been adopted. This may be stated briefly as follows:

In the absence of any sign of infection penetrating joint wounds made by sharp instruments, supposedly fairly clean, may be treated expectantly with sterile dressings. This applies also to perforating wounds made by bullets of high velocity, especially in the absence of fracture. They must be watched carefully, however, and must be opened up at the first sign of an infection. Other wounds demand operation.

THE OPERATION OF DEBRIDEMENT, as practised so extensively, aims to remove not only the foreign body but also its infected track through the tissues. In wounds of the soft parts all devitalized tissue in the neighborhood is

also excised, but this feature is not so prominent in operations on joints. The wound of entrance is excised down into the joint, and the foreign body is removed. If the projectile is imbedded in bone or soft tissue, this is removed with it. The track of the missile is excised, with chisel and gouge, with a curette only if necessary. All foreign bodies, such as shreds of clothing, and all loose fragments of bone are removed. If the joint has been so badly damaged that useful function is not to be expected, resection had best be done forthwith.

After lavage of the joint cavity with normal salt solution, the capsule is sutured, and the wound of the skin also, unless its infection is suspected. If so, the wound in the skin is left open. Some surgeons recommend filling the joint cavity two or three times with ether after lavage with normal salt solution. If the joint fills with fluid thereafter, it should be aspirated. No drains should be left in the joint. Early active and passive motion, as soon as the wound has healed, are very important.

In this procedure one must always avoid infecting sterile tissue. Instruments which have touched infected tissue should be discarded for fresh ones, and the gloves must be changed as often as necessary.

In the early stage of infected wounds the same treatment may be carried out tentatively, perhaps modified by drainage for a day or two. Payr maintains that in joint wounds without bone damage, the infection is often superficial in the synovial membrane, and can be controlled by irrigation (injection), with or without temporary drainage, either simply through small openings in the capsule, or with a glass tube.

With frank infections, after thorough debridement and cleansing, drainage of course is necessary. Willems

advises, in addition to the openings in the capsule, early active motion to accomplish drainage. Opinions differ as to the efficacy of this treatment. The Carrel-Dakin treatment has its advocates. Whatever method of drainage be adopted, care must be taken that it is thorough. It is not enough simply to make openings in the joint unless they actually drain it. If necessary, bone must be removed, and no recess must be left for the accumulation of pus. Some surgeons advise resection, others have been disappointed with the results of the operation. Amputation often will be necessary.

CHAPTER X

HÆMOPHILIAC JOINTS

HEMORRHAGE into the joint, especially into the knee, is a fairly frequent occurrence in that interesting condition known as the hemorrhagic diathesis or hæmophilia. It is observed much more often in men than in women, as is the diathesis to which it is due. The tendency to bleed uncontrollably at the slightest scratch is hereditary, and is handed down through the female element. Addis¹ investigated a number of these families of "bleeders" and has added much to our knowledge of the subject of hæmophilia. It is important to know the manifestations of the diathesis in the joints in order to avoid making a blunder that may be fatal.

As the result of a slight trauma, or, as some claim, without trauma, a joint of a bleeder, usually the knee joint, swells up, "like a balloon," as one patient put it. Pain as a rule is not a prominent symptom; the chief complaint is the interference with function from the presence of the fluid. Fluid can be detected in the joint, and sometimes a soft

¹ ADDIS, T.: "The effect of the administration of calcium salts and of citric acid on the calcium content and coagulation time of the blood." *Quart. J. Med.* 1908-09, v, 2.

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crepitation. The synovial membrane proliferates, sometimes enormously, and its internal surface may become

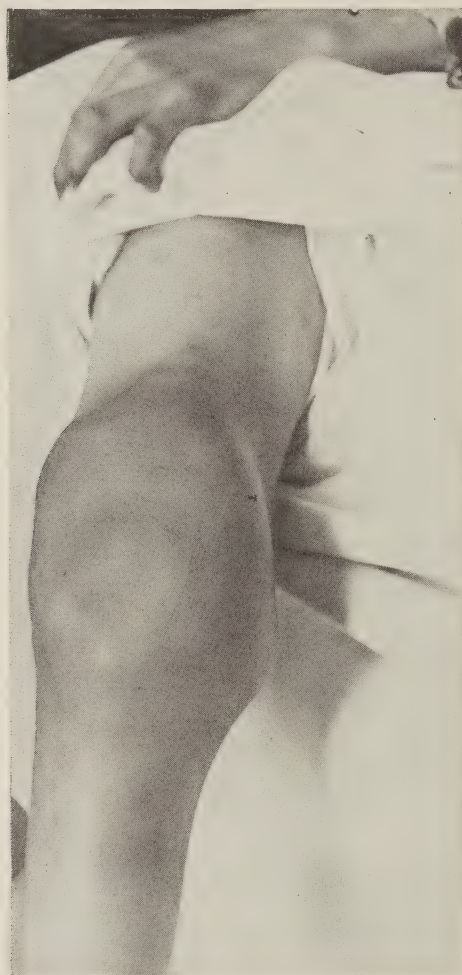


FIG. 40.—Hæmarthrosis of the knee in a "bleeder."

shaggy, with the enlarged villi branching like moss on a rock.

The fluid may slowly be absorbed and the joint may return to normal, to be again attacked at a later period.

Again layers of fibrin may be deposited upon the synovial membrane and possibly upon the cartilage. These layers of fibrin may become organized, and may give rise to dense adhesions, which cause a practically complete ankylosis.

A sudden, fluctuating, comparatively painless swelling of a joint without history of injury, in the absence of physical signs of tabes, should always awaken the suspicion of hamarthrosis, and should occasion a search for a family

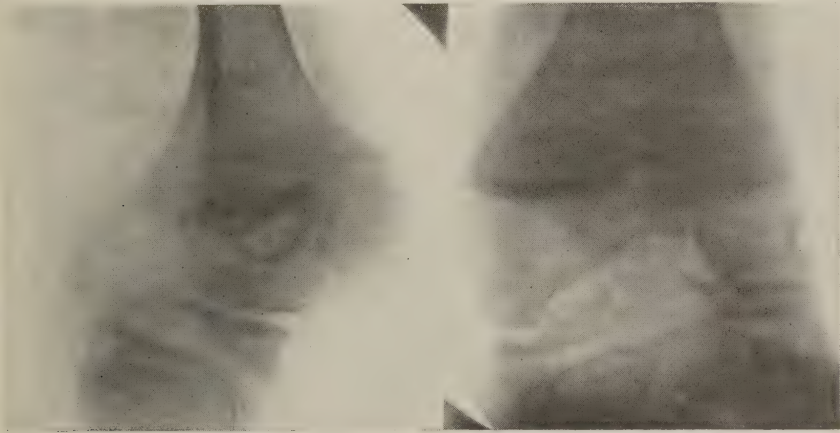


FIG. 41.—Skiagram of a case of old fibrous ankylosis in a "bleeder."

history of bleeders, and for a personal history of profuse hemorrhage after slight injury.

FOR THE TREATMENT of the acute attack, rest, pressure and cold applications are probably best. After the hemorrhage has ceased and a little time has elapsed, hot applications and gentle massage may possibly promote the absorption of the exudate. In case of need, blood transfusion may be done, or blood plasma may be injected.² The advantage of any such procedure is temporary only;

² Personal note of Dr. Addis: "Hot applications and gentle massage I should be afraid to use. I should prefer temporary immobilization with pressure.

"Blood transfusion should only be used in case of danger to life. This would not arise in a hemorrhage confined to the joints."

the coagulation time is shortened, but later is lengthened again, so that the patient is in greater danger than he was before.

It is hardly necessary to emphasize the importance of avoiding in all circumstances and in every stage, any operative interference with a joint hemorrhage in a bleeder.

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SECTION III.

CHRONIC OSTEOMYELITIS

CHAPTER I

PHOSPHORUS NECROSIS

INDIRECTLY, and consequent upon the changes in the bone produced by phosphorus, bacteria from the mouth may cause a chronic suppurative osteomyelitis in the maxilla or more often in the mandible. The disease is usually found in workers in phosphorus match factories, and especially among those who have bad teeth. With the introduction of sanitary precautions it has grown rare.

Hand in hand with the suppuration and necrosis in the bone and in the marrow, goes the production of new bone not only in the periosteum, but sometimes in the bone itself. The teeth loosen and fall out, and the soft tissues become infiltrated and inflamed. The entire mandible may be killed, and its articulation may become ankylosed. Death often results. After the removal of the mandible a new bone may be formed by bone production in the periosteum.

The treatment is prophylactic and operative. The mouth and teeth of match-makers should be kept scrupulously clean and in good condition. Factory employés should not eat nor drink in their work-rooms, and by attention to their hands and to their finger nails should avoid carrying any phosphorus into their mouths. Work-rooms must be kept clean and well-ventilated. Where proper precautions are observed, phosphorus necrosis will rarely be seen.

The operative treatment consists in the resection of the diseased bone. The resection should be a free one, and all the affected tissue must be removed. As a rule new bone will be built up to replace that which is removed.

SCLEROSING OSTEOMYELITIS

A rather rare form of osteomyelitis, the so-called sclerosing osteomyelitis, has been described which has an acute or subacute onset and does not lead to the immediate formation of pus. More or less of the bone becomes very dense and thickened. Sometimes small abscesses with sequestra are found in this mass of dense bone. Whether this disease should be included under the head of acute or of chronic osteomyelitis is doubtful. The treatment consists of eradicating any possible focus of infection in the body, and cleaning out any abscesses with the surrounding sclerosed bone.

Another rather bland form of chronic osteomyelitis is occasionally seen in workers in mother-of-pearl. It has no tendency to suppurate, and yields readily to the removal of its cause.

CHAPTER II

SYPHILIS

THE bone manifestations of syphilis are very frequent. Not only is the infection carried to the bone marrow at an early period, but it remains there almost indefinitely. It manifests itself in the secondary and in the tertiary stages, and also after the subsidence of the active period of the disease in the form of the so-called "neurotrophic" bone lesions.

Congenital bone syphilis is fairly frequent, in fact its lesions are among the most characteristic in the body. It has been pointed out that congenital syphilis is really acquired syphilis, acquired *in utero*, and that its peculiarities are due simply to the differences in foetal and infantile tissues from those of the adult. Hereditary syphilis probably is a misnomer, as implying an infection of the germ plasm.

In studying syphilis of the bones and joints one is struck by the marked, and, at first sight, unaccountable difference of opinion that prevails on most phases of the subject, but the difference of opinion is not so strange as it seems. Until the last few years the cause of syphilis had not been identified. Therefore the diagnosis rested upon the history, upon the presence of other known (or presumptive) evidences of syphilis, upon the therapeutic test, or upon all three. Even to-day the vast majority of clinical diagnoses is made without the demonstration of the spirocheta pallidum, and in such cases the pathologist never receives any of the material for examination, for no operation is done.

On the other hand the pathologist regularly performs necropsies upon the bodies of still-born fœtuses and of young infants, in which he can demonstrate syphilis. With these cases the clinician is not brought into contact. The morbid processes observed by the pathologist and by the clinician appear to be entirely different, but in point of fact they are quite the same, and differ only in location and details.

PATHOLOGY.—The characteristic lesion of syphilis in bone is a proliferative inflammation in the marrow—a syphiloma, a gumma. This may occur in any part of the bone, though its situation is largely determined by the age of the patient. In congenital syphilis, as seen in infants and children, the ends of the long bones and the shaft of the bones of the hands suffer most; in the adult the shaft of the long bones is most often attacked. The resulting process depends largely upon the location. While certain peculiar types of osseous syphilis can be recognized as more or less standard, it is necessary to remember that they are by no means invariable, and that here as elsewhere in the body, the disease often manifests itself in most unusual ways.

EARLY CONGENITAL SYPHILIS

Although a few cases of congenital bone syphilis had been published, the disease was considered to be very rare, in infants and children, until the publication of Wegner's article in 1870.¹ Then quickly the investigations of Parrot² and of Waldeyer and Koebner,³ in 1872, and of

¹ WEGNER, GEORGE: "Ueber hereditære Knochensyphilis bei jungen Kindern." *Archiv. f. Path. Anat. u. f. klin. Med.*, 1870, i, 305.

² PARROT, M. J.: "Sur une pseudo-paralyse . . . de syphilis héréditaire." *Archives. d. Physiol. Normale e. Path.*, 1872, iv, 319, 470, 612.

³ WALDEYER u. KOEBNER: "Beiträge zur Kenntnis der hereditären Knochensyphilis." *Arch. f. Path. Anat.* 1872, lv, 367.

Taylor,⁴ in 1874, established the fact that bone lesions in early congenital syphilis, so far from being infrequent, were among the most common manifestations of the disease.

The peculiar lesion of congenital syphilis, the "osteochondritis syphilitica" of Parrot is located immediately adjacent to the epiphysial cartilage on its shaft side, that is, in and near the zone of provisional calcification. It consists essentially in a syphilitic myelitis. The new granulation tissue in the marrow breaks up the regular formation of bone. Under the microscope one sees tongues of it pushing up into the epiphysial cartilage. The columns of calcified and calcifying cartilage matrix in the zone of provisional calcification are, to a great extent, absorbed, so that the zone or a part of it may eventually consist of little else than granulation tissue, separating the epiphysis partially or completely from the shaft. Later the granulation tissue undergoes necrosis.

The zone of provisional calcification becomes much wider than normal and appears as a fairly broad white or pinkish band, irregular in its outline, instead of as a thin, barely perceptible line. Later the tissue breaks down and becomes a mass of grayish-white, brittle, mortar-like material, either in a small area or running across the entire width of the bone and possibly separating the epiphysis from the shaft. The process in the marrow is not regular and sharply defined, but decidedly irregular, wavy, and notched. Vessels from the marrow push up into the epiphysial cartilage. The resulting irregular outline of the shaft side of the epiphysial cartilage, somewhat similar to that in rickets, is perceptible to the naked eye and appears also in the X-ray plate. The X-ray shows also a

⁴ TAYLOR, R. W.: "Syphilitic lesions of the osseous system in infants and young children, etc." *Am. J. Obstet.*, 1874, vii, 53, 177, 559.

rarefaction of the bone corresponding to the irregular area of the disease, on the shaft side of the epiphysial cartilage. The broken down syphilitic granuloma may later break through the periosteum and communicate with the surface and become secondarily infected, or resolution may take place under appropriate treatment. If the epiphysis have been separated, union usually is brought about by new bone laid down in and under the periosteum, and the growth of the limb is not retarded.

The changes described above are often seen in still-born syphilitic foetuses. They may, however, be present in syphilitic children born alive or may appear during the first few months of life. They occasion swellings in the bone in the neighborhood of the joint, sometimes quite painful and sensitive to pressure, sometimes not so painful. The resulting condition is what Parrot described as "pseudo-paralysis." The limb hangs limply. The child refuses to move it, and cries when it is moved. With separation of the epiphysis, a false point of motion can be found, with a soft crepitus. When union has been brought about, the thickened periosteal bone, shaftward from the epiphysis can be felt.

The gross changes in and about the epiphysis are somewhat similar to those seen in rickets, and the similarity is so great that certain observers in the past have thought that syphilis was the cause of rickets. The essential points in the diagnosis are: first, syphilis appears at an earlier age than rickets; second, with syphilis other marks of the disease such as roseola, mucous patches, etc., are usually present, possibly also other changes in the shafts of the long bones and the skull; third, the changes about the epiphysis in syphilis usually, as far as clinical evidence goes, are single or at best are evident in only two or three

places, whereas with rickets they are more or less general; fourth, syphilis is more painful than rickets.

The changes in the region of the epiphysis in scurvy, are somewhat similar clinically to those of syphilis, but scurvy usually appears somewhat later, the irregular appearance of the zone of calcification and the bone absorption do not appear in the X-ray plate in scurvy, the onset is sudden, the pain perhaps is more severe, other signs of syphilis are absent, and signs of scurvy, such as hemorrhages from the gums are present.

Besides this so-called syphilitic osteochondritis another notable marrow change is often met with in young syphilitic children according to Wegner, namely fatty degeneration of the vessels and cells of the marrow. This may be more or less diffuse, or may be circumscribed, and gives to the bone marrow a yellowish or pinkish yellow color. Perhaps to these extensive marrow changes is due the well known anæmia of syphilitic children.

In older children three syphilitic lesions are often found in the bones of the extremities: first, cortical changes similar to those of the adult, later to be described; second, disease of the bones of the hand, most often of the proximal phalanx, similar to the spina ventosa of tuberculosis; third, disease of the ends of the long bones with an accompanying arthritis.

Syphilitic disease of the marrow of the bones of the hand is fairly frequent in children. It causes a rarefaction of the bone and an enlargement, more or less general, but especially marked at its proximal extremity. It shows little or no tendency to involve the joint, and quite often breaks through the cortex and the periosteum, communicates with the surface, becomes secondarily infected, and leads to the establishment of persistent sinuses. The

irritation of the disease in the marrow with the resulting destruction of bone in the interior, is said to cause the formation of new bone in the periosteum, perforated in one or more places for the discharge of the necrotic material within. A probe passed through one of these holes in the shell of the bone easily enters the large cavity.

This syphilitic dactylitis is as a rule not very painful. The swelling may be slight but usually is quite well marked, giving the bone twice or thrice its normal diameter. The length of the bone may also be slightly increased.



FIG. 42.—Dactylitis, probably syphilitic but treated for a long time as tuberculosis.

The general shape of the bone in its breadth is round, while it appears to have in its longitudinal direction an oval shape (Taylor). It is somewhat broader at its base than at its distal end, and the swelling begins quite perceptibly at the metacarpo-phalangeal joint, which it enlarges, and ends somewhat abruptly at the next joint, that is, of course, with disease of the proximal phalanx, the usual lesion. The integument is usually stretched and may or may not be reddened.

The Röntgen rays show the irregular structure of the bone, and its enlargement, especially at the proximal end. This peculiar shape of the bone may help in the differentia-

tion of syphilitic from tuberculous dactylitis, as may the presence of other syphilitic lesions in the body, and perhaps the reaction to anti-syphilitic treatment, but very often the problem cannot be solved without the aid of the microscope and the guinea-pig test.

With syphilitic involvement of the marrow in the ends of the long bones in children a pathological process results in the bone and joint so similar to that of tuberculosis that the clinical differentiation may be extremely difficult or even impossible. Not only are the essentials of the pathological process the same, but also the symptomatology and physical signs. The realization of this fact alone will save one from humiliating mistakes. The differential diagnosis will be taken up under the head of tuberculosis.

One of the best known evidences of congenital syphilis in the child is the so-called "saber shin," an anterior bowing of the tibia probably caused by a chronic syphilitic process in the marrow in the superficial part of the cortex. It gives to the child's legs a characteristic appearance, not to be forgotten when once seen, and hardly to be confused with the outward bend of the ordinary bowlegs.

BONE SYPHILIS IN THE ADULT

Syphilitic osteomyelitis is frequent in the adult. The gummatous inflammation causes primarily, whatever its situation, a rarefaction, an absorption of the bone, and, if the process be very severe, death of the bone to a greater or less extent. The tissue breaks down and forms jelly-like or mucilaginous or cheesy masses, which may be absorbed under appropriate treatment, may be encapsulated, or may become secondarily infected and lead to the formation of sinuses.

Probably this syphilitic inflammation can exist in any

part of the bone marrow, but it occurs in an overwhelming proportion in the superficial part of the cortex directly under the periosteum. The bone becomes spongy and later sclerosed in that locality, and new bone is produced in and under the periosteum. This new bone production dominates the clinical picture and gives the entire lesion its stamp, and it is customary to allude to this form of syphilis as a periostitis, and to regard bone production as the essential factor. The great weight of authority is in favor of this view, but I believe it is an error, and consider the sequence to be: first, proliferative inflammation in the marrow; second, bone destruction; and third, bone formation. This so-called syphilitic periostitis probably may occur almost anywhere in the body, but its favorite location in the extremities, is in the shafts of the long bones, especially in that of the tibia. It may appear in the form of a more or less circumscribed swelling, or in a more diffuse and irregular thickening of the entire shaft or of a portion of it. Again the new periosteal bone may be present in small patches.

This cortical lesion possibly may occur in any period of congenital syphilis, but is considered more or less characteristic of the tertiary stages of the acquired form. It may be painless or comparatively so, but usually is quite painful; in fact the boring pains of this form of bone syphilis, with their nocturnal aggravation, are notorious. Local sensitiveness is usually present. The overlying tissues may or may not be inflamed. If the gumma break down and become secondarily infected, a sinus is formed which may persist indefinitely, and in a general way may be said to resist treatment in proportion to the length of time it has existed. The openings of these sinuses are wont to be dark red, dirty, sluggish, undermined and ragged, not pale, puffy and pouting, like the tuberculosis sinus.

X-RAY DIAGNOSIS.—Syphilis usually involves more than one bone; osteomyelitis usually only one bone. In syphilis there is rarely the extensive demineralization (so-called “bone atrophy”) which is a prominent feature of osteomyelitis.

Syphilis involves especially the superficial part of the cortex, the only medullary involvement usually being by a smooth narrowing of the medullary canal, due to the thickening of the cortex at the expense of the medulla; whereas osteomyelitis begins as an acute myelitis with secondary periostitis, of a less regular type.

These points apply to active cases alone. Old lesions are frequently impossible to differentiate.

Bone forming sarcoma of the periosteal type may be so characteristic as to be in little danger of confusion. The fungoid, “hair on end” appearance (“whiskers”), due to the vertically disposed bone “rays” serves to differentiate from other proliferations. Bone forming sarcoma produces enlargement of the bone, which is locally more extensive than the enlargement of lues or osteomyelitis, but which does not extend so far up or down the shaft. Also there is much less proliferation in comparison with the amount of enlargement.

It is necessary to say that the X-ray diagnosis is suggestive, not final. I have seen a mixed series of cases thrown on the screen whose nature defied detection.

Syphilitic disease of the marrow of the spongy bone of the spine and extremities, is not nearly so frequent as the preceding, that is as far as one can judge from clinical evidence. It causes a diffuse and more or less irregular absorption of the bone, as revealed by the Röntgen rays, very like that caused by tuberculosis, and *sometimes not to be distinguished from it by clinical examination*. These lesions in the spongy bone do not show a marked tendency

to break down, are usually slow and chronic, are often painless, and, unlike tuberculosis, may exist indefinitely in the immediate neighborhood of the joint without involving it.

The X-ray picture is similar to that of the members of the first great type of arthritis, for new bone production



FIG. 43.—Bone syphilis. The patient of whose radius this is a picture had been treated for a long time for tuberculosis, and recovered under appropriate treatment.

in the periosteum is not always to be found in the neighborhood of the joint in this form of syphilitic myelitis. The diagnosis is made by the history, by the detection of other signs of syphilis in the body, especially new periosteal bone in other locations, by the Wassermann and Noguchi reactions, and, of greatest importance, by the results of anti-syphilitic treatment. It is a safe rule to regard every case of suspected tuberculosis as possible syphilis until syphilis has been definitely ruled out. On the other hand, the rare

cases of shaft tuberculosis are usually mistaken for syphilis. Radiologically the two are the same.

A third and much rarer form of bone syphilis is a gumma of the central marrow canal. This usually results in the formation of a more or less circumscribed collection of mucilaginous material in the medulla, about which the bone may or may not be sclerosed. This lesion, as a rule, is not very painful, but the reaction in the periosteum over the portion of the bone where it is situated causes a certain degree of local sensitiveness. The Röntgen picture may be quite similar to that of a bone cyst, but careful observation usually detects new bone production in the periosteum over the gumma, whereas this bone production is absent in the ordinary bone cyst.

Besides the syphilitic arthritis resulting from disease in the marrow of the neighboring bone, another form of syphilitic arthritis is fairly frequent in the tertiary stages of the dis-

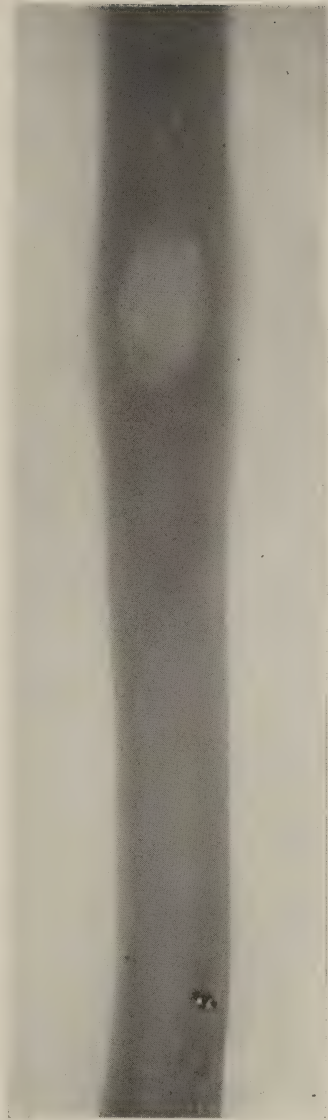


FIG. 44.—Syphilis of the shaft of the humerus. This case was operated on with an erroneous diagnosis of bone cyst. Note the reaction in the periosteum, which should have made a correct diagnosis easy.

ease. It is essentially a proliferative inflammation of the synovial membrane—a syphilitic synovitis—and shows little or no tendency at any stage to involve the bone marrow. The synovial membrane proliferates and becomes villous, and pours out a secretion which fills the joint. This syphilitic synovitis may be uniarticular or multiarticular, affects by preference one or both knees, is usually practically painless, and responds promptly to treatment. The skiagram is negative except for the swelling of the soft parts.

Tabetic joints and bone lesions are an interesting manifestation of late syphilis. They are probably degenerations rather than inflammations. Whether or not they should be included in a book on bone and joint inflammation, is doubtful.

TREATMENT.—This, in its main features, is that of syphilitic lesions anywhere in the body, namely by arsenamin, mercury and the iodides. The restoration of the normal structure of an apparently hopelessly damaged bone or joint is sometimes remarkable, but when secondary infection has taken place the treatment must often be continued for a long time. Of course operations should be avoided if possible, and this fact is well known. The tendency of clean wounds to break down after operations on syphilitic bones is also well known. A peculiar fact, or possibly a theory, in the treatment of syphilitic arthritis is the lack of response to immobilization of the joint, and this fact is not without value from a diagnostic standpoint. If a syphilitic joint be immobilized, pain does not decrease as a rule. Indeed, in contrast to tuberculosis, it may grow worse, and necessitate the removal of the dressing.

CHAPTER III

CHRONIC OSTEOMYELITIS OF UNKNOWN ORIGIN

UNDER this heading can be included a number of clinical forms of marrow inflammation whose identity is not firmly established, and whose differentiation is by no means definite or exact. Indeed that they are all marrow inflammations is not universally acknowledged. Three of them may be grouped. They possess in common certain pathological features, some of which may be regarded as an exaggeration of those ordinarily observed in the bones with the advancement of age. They are probably closely related, and probably are variations of the same process. They are Paget's disease, or *ostitis deformans*, *leontiasis ossea*, and the so-called *ostitis fibrosa*. Even to find a name under which to group them is not easy, but for convenience, we will call the group

OSTEOMYELITIS FIBROSA

The first and probably the fundamental change in the members of this group is a fibrosis of the marrow. With this goes an irregular production and absorption of bone, with or without the formation of osteoid tissue. Cyst formation is also a frequent accompaniment. These characteristics, as we shall see, are shared by the second great type of arthritis, and some writers with good reason have regarded the two diseases as essentially variations of the same pathological process. Others deny any relation between them.

INFLAMMATION IN BONES AND JOINTS

PAGET'S DEFORMING OSTEOMYELITIS, OSTITIS DEFORMANS



FIG. 45.—Paget's deforming osteomyelitis;
Stanford Clinic Case No. 1.

This disease was first established as a clinical entity by Sir James Paget, who, in a paper read before the Royal Medical and Chirurgical Society, placed five cases of it on record in such detail that little definite has since been added to his description.¹ Five years later he published seven more cases.² The disease is comparatively rare, though probably not as rare as is generally thought. Four or five cases are on record at Stanford.

ÆTIOLOGY. — Nothing definite is known as to this. The whole appearance of bone and marrow is that of a low grade chronic infection, though no definite proof of an infection ever has been adduced. The theory that the disease was a manifestation of syphilis has been advocated, but has been

¹ PAGET, JAMES: "On a form of chronic inflammation of bones." *Medico-chirurgical transactions*, 1877, lx, 37.

² PAGET, JAMES: "Additional cases of ostitis deformans." *Med. Chir. Trans.*, 1882, lxxv, 225.

almost universally discarded. Several of Paget's cases died of malignant disease, and Paget thought that there was same causal relation between the two, but other observers have not confirmed his observation. The similarity of



FIG. 46.—Paget's deforming osteomyelitis; Stanford Clinic Case No. 2.

Paget's disease to the second great type of chronic arthritis might suggest the alveolar processes of the jaws as a possible atrium of infection.

Paget's disease is essentially a disease of middle and later life, though one case was observed at the age of

twenty-one. It is most commonly seen after forty, and the majority of patients have been men. A slight familial tendency has been noted.

The bones most frequently and most severely affected in Paget's disease are the tibia, the femur, the calvarium,



FIG. 47.—Paget's deforming osteomyelitis. Photograph of one of Doctor Ethan Smith's patients.

the clavicle, and the spine. Apparently almost all the bones of the body may be involved except those of the face, which always escape.

The disease begins in the superficial part of the bone cortex. Probably the initial macroscopic change is a rarefaction of the bone in this vicinity, a rarefaction which

slowly advances inward. An irregular production of osteoid tissue and new bone, especially new periosteal bone, follows this, overshadows the initial rarefaction and clinically is by far the most prominent feature of the disease. The bones become thickened and deformed, and, composed as they are

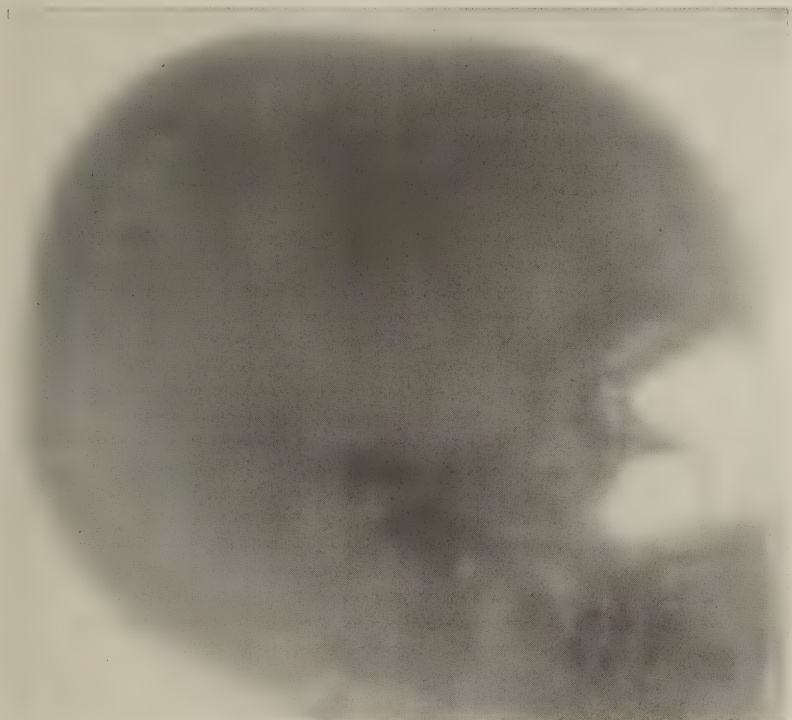


FIG. 48.—Paget's deforming osteomyelitis. Skiagram of the skull

of poorly formed bone tissue, yield to pressure and to strain. The head sinks between the shoulders, the clavicles become prominent, the spine bows and sinks together, and the femora and the tibiae bow outward. The skull may become enormously enlarged, sometimes with a smooth surface and sometimes with irregular nodules. As the result of the shortened trunk and the bowed extremities the arms appear disproportionately long. This gives to

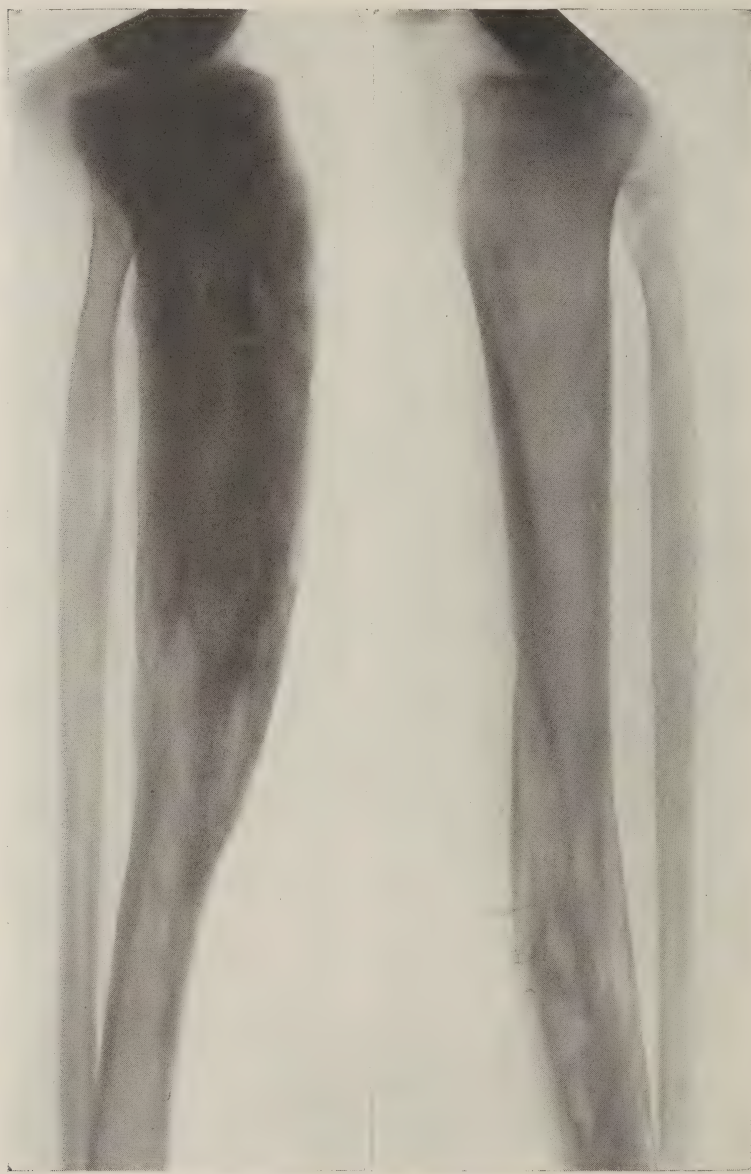


FIG. 49.—Paget's deforming osteomyelitis. Skiagram of the bones of the legs.

the patient an appearance which has been well likened to that of an anthropoid ape.

HISTOLOGY.—The marrow consists of a vascular connective tissue, more or less rich in cells. Typical giant cells are often seen, sometimes in great numbers, and giant cell tumors are not rare in this disease. The Haversian canals are widened, and are filled with vascular tissue, especially in the superficial portions of the bone. The whole

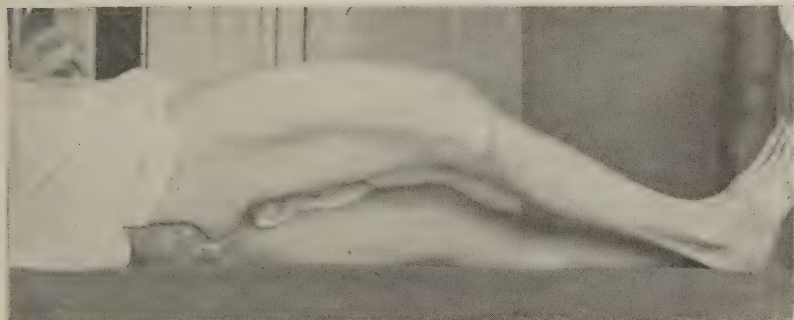


FIG. 50.—Paget's deforming osteomyelitis—Doctor Ethan H. Smith's second case. This photograph was taken in 1906, and the disease then was presumably of about twenty years standing. Eight years later the femur was fractured at about the junction of its proximal and middle third. The fracture did not unite. Doctor Smith amputated about five months later. He "pinched the neck of the femur off" between his thumb and forefinger. The bone must have been very soft. The next figure shows the specimen.

architecture of the bone is changed. The Haversian systems as such are obliterated, the cortex is thickened and the central marrow canals or the diploë may be obliterated by the overgrowth of new bone and osteoid tissue. Sometimes the new bone is of normal consistency, sometimes it cuts easily with a knife. At times greater or smaller islands of sclerosed bone tissue are formed in the midst of the other bone. The osteoid tissue evidently may be ossified later, or at times may itself fall a victim to absorption. The margins of the trabeculæ often show the classic picture of rarefying osteitis—so-called osteoclasts in Howship's lacunæ. Cysts have been observed in some cases. In

Paget's cases "the medullary structures appeared to the naked eye as little changed as the periosteum."

Strange to say, in spite of all the bone absorption, fractures do not often occur in Paget's disease.

SYMPTOMATOLOGY.—Pain may or may not be present. As a rule it is not severe. Frequently the first thing



FIG. 51.—Distal two-thirds of femur, and proximal end of tibia of Doctor Smith's second case, sawn sagittally. Note the disorganization of the extreme distal end of the femur, and the complete change from the normal architecture of the whole bone. The cortex cut easily with the knife in the laboratory. It consisted of rather open-meshed fibrous tissue containing scattered, slender bone trabeculae.

noticed by the patient is that he is compelled to wear a larger hat than formerly, and must increase its size as time goes on. When the bones of the lower extremity are first involved, then their bowing will be the first thing noticed. The X-ray picture will show a marked thickening of the affected bones, usually more or less irregular in structure. Border line cases may sometimes be hard to distinguish, but the typical case of Paget's deforming osteomyelitis is easily recognized. The enlarged cranium, the bowed and

shortened spine, the bowed lower extremities, the short neck and the ape-like build present a picture not easily confused with anything else. The disease is probably not as rare as has been thought, and escapes recognition only when one is not alive to the possibility of its occurrence.

TREATMENT.—No treatment has ever been accepted for this disease. On the theory that the appearance of the marrow indicates a low-grade chronic infection as a causal factor, a search should be made for a focus of infection anywhere in the body, especially in the teeth. If one be found, it should be removed. Following out what at present is little more than a theory, it will be well to search for the amœba in the intestinal canal.

LEONTIASIS OSSEA

The pathological features of this disease are similar to those of the preceding. Its distinguishing characteristic, however, is a marked involvement of the bones of the face, giving to the patient the appearance of one with leprosy. It is of very infrequent occurrence. Besides the ordinary symptoms of pain and discomfort, the involvement of the facial bones may cause severe disturbance in the organs of special sense.

Nothing is known as to the cause of leontiasis ossea, nor as to its treatment. On the chance that it may be caused by some obscure source of infection, any focus of possible infection should be sought and removed, especially in the jaws.

OSTEOMYELITIS FIBROSA, OTITIS FIBROSA

Without the peculiarities of the two last named diseases, a form of chronic osteomyelitis occasionally is seen, characterized by the change of the marrow to fibrous tissue. Giant cells are frequent in this fibrous tissue. The disease

may be generalized or local. Cysts may form in the fibrous tissue, and the architecture of the bone is usually changed. The bone may be distended, but the thickened cortex and the periosteal reaction observed in syphilitic disease is absent. Pain may or may not be present. Fracture occasionally occurs. The diagnosis is made with the Röntgen rays.

The cause of this disease also is not known, but it is probably some obscure form of infection, possibly in the jaws. The tendency of the marrow to become fibrous as age advances, is to be borne in mind.

TREATMENT.—Any focus of infection should be removed. Cysts may be cleaned out by operation.

Giant cell growths in bone, the so-called giant cell sarcomata or benign myelomata are quite similar to the preceding, are probably inflammatory, and should be classified with the chronic inflammation, but as they are usually regarded as new growths, they will be omitted from consideration.

PULMONARY HYPERTROPHIC OSTEOARTHROPATHY

Somewhat akin to the preceding diseases is a rather rare condition first described by Bamberger, and later, by Marie, whose bone manifestations apparently consist of a chronic osteomyelitis with a production of new periosteal bone. The name is a most unfortunate one in every respect, but until we know more about the disease, it is hardly worth while to change its name. In the first place, it is caused by other things than pulmonary lesions, and in the second place the disease apparently may exist without any bone or joint involvement whatever.

ÆTIOLOGY.—Four groups of causes may be enumerated:

1. Suppurative or gangrenous processes in the lungs or pleura.

2. Infectious diseases.

3. Valvular heart lesions, especially congenital.

4. Malignant tumors in the lung or in the mediastinum.

SYMPTOMATOLOGY.—What has always been considered the distinguishing mark of the disease is a clubbing of the terminal phalanges of the fingers (the Trommelschlegelfinger of the Germans), together with a curving or beak-like deformity of the nails. The fingers are wont to be cyanosed as well. The swelling of the end phalanges is entirely in the soft parts: The bone is not affected.

Associated with this peculiar deformity may be a thickening of the rest of the fingers, and sometimes also of the forearm (or leg) as well. Examination shows that this thickening is due to new bone formation, which is plainly shown by the X-rays to be located on the outside of the cortex.

TREATMENT.—This consists in the removal of the cause if possible. Definite improvement has been reported from successful treatment of the causal lesion.

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CHAPTER IV

RICKETS, RHACHITIS

RICKETS is a constitutional disease of infancy and early childhood, whose chief anatomical changes appear in the bones. Good authority is behind the statement that the disease occasionally occurs in foetal life. Certainly its manifestations have been observed in very early infancy. The so-called "late rickets" of adolescents is probably a misnomer.

ÆTIOLOGY.—Nothing definite is known as to this. The marrow changes point strongly toward chronic infection as a cause, and various investigators have claimed to have produced the disease experimentally by the use of infectious agents, but their claims have not been substantiated. On account of the similarity of the bone changes in rickets and in syphilis, syphilis has been considered as a cause of rickets, but the two diseases are quite distinct. One does not cause the other.

The cause of rickets is closely bound up with the processes of nutrition. The disease can be produced experimentally in animals, by surrounding them by unnatural conditions. The essential element of these unnatural conditions is popularly thought to be an error in diet, an absence from the food of some essential element, but Leonard Findlay and others stress the importance of confinement, and of the lack of fresh air. Perhaps all are but contributing causes.

Rickets is more common in certain races than in others, and in certain climates than in others. People from

southern climates, transplanted to northern, are especially liable to the disease, possibly because they live more in-doors in their new domicile. On the other hand the Esquimaux are said to be exempt from the disease. It is much more frequent in the city than in the country. It is common in England and in Germany, and in our northern cities many severe cases are seen among negroes and Italians.

PATHOLOGY.—The bone changes alone interest us here, and it is hard to say which is the fundamental change. The

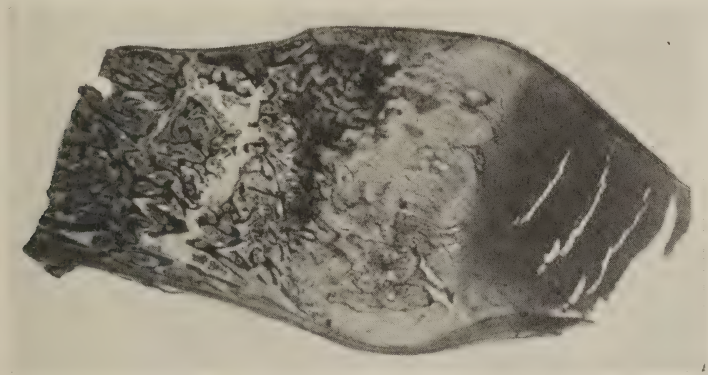


FIG. 52.—Rickets. Low power photomicrograph of the costochondral junction. Note the irregularity of outline of the cartilaginous disc on its left border.

marrow becomes very vascular and undergoes a fibrous change. Islands of osteoid tissue appear in it, and the Haversian canals widen. In other words the marrow is the seat of a chronic inflammation. The most marked evidences of the disease are on the outside of the cortex, and in the region of the epiphysial disc; in other words, in those parts of the bone where growth is most active.

The changes at the epiphysial line (as at the costochondral junction) are among the most characteristic. Masses of marrow tissue push in among the columns of cartilage cells in the zone of provisional calcification (Nichols). The zone of provisional calcification disap-

pears, the cartilage cells lose their typical arrangement, and the epiphysial disc becomes irregular and decidedly broadened. On its diaphyseal side masses of osteoid tissue instead of bone, are laid down, and in this osteoid tissue islands of cartilage persist. In other words ossification is



FIG. 53.—Skeleton of rickety child.

halted. Neither calcification nor ossification takes place. Layers of osteoid tissue form on the trabeculae of the diaphysis, but whether or not there is ever a reversion of bone tissue to osteoid is doubtful. Probably the process is best interpreted as a failure to build up bone, rather than an actual tearing down of bone already built up, aside from the tearing down inseparable from the remodelling incidental to growth.

The periosteum is thickened, and layers and arches of osteoid tissue instead of true bone are formed in it. Rarefaction of the cortex is proceeding meanwhile, and the Haversian canals are widened and contain vascular marrow. The masses of osteoid tissue in the periosteum give to the bone a thickened appearance. They are more prominent in certain situations than in others, especially upon the parietal eminences of the skull. In other parts of the skull absence of bone formation, as well as of osteoid tissue, is the prominent feature. This is best seen in the occipital

bone, the so-called craniotabes. The bone is thin and parchment like, easily indented by pressure with the finger. This craniotabes is one of the most important diagnostic signs of rickets.

Lack of bone formation in the cranial bones causes a persistence of the fontanelles, also an important diagnostic sign.

In gross the bones become soft and bent, and are broadened in the region of their epiphyses. Fractures are said to be frequent. Vascular, soft, spongy, grayish-red masses of tissue are seen in the medulla and on the cortex. The epiphysial line is broadened, and, instead of being sharply defined and approximately straight, is irregular. Medullary and osteoid tissue extends into it. The central medullary canal is widened.

When the disease has run its course, calcification and ossification of the osteoid tissue take place, and the bones become denser than normal—sclerosed. The deformities may persist or they may disappear. Almost always in later life, the enlarged epiphyses and the peculiar shape of the head, remain as vestiges of the infantile disease.

SYMPTOMATOLOGY.—The constitutional manifestations of rickets are numerous and well known. Among them are the nervous symptoms, the convulsions, the laryngismus stridulus, the restlessness at night, etc. The patient is anæmic and pasty looking, and is afflicted with sweating about the head. Usually he is pot-bellied and suffers from constipation. Teething is late and the child does not begin to walk at the usual time.

All these are quite important, but they do not justify a diagnosis of rickets. This is only to be made upon the bone changes. The two most important of these perhaps are the craniotabes and the epiphysial changes, the latter

including the changes at the junction of the ribs with their cartilage.

Craniotabes is a very early sign, and by some is considered as an invariable and pathognomonic sign of rickets, provided its presence is carefully sought. It may be more

or less general in the occipital bone, or small areas only may be involved.

The enlarged epiphyses are easily detected. The enlargement of those of the wrist and ankle is most evident. The enlargement of the costo-chondral junctions causes a line of knobby prominences on the chest wall, known as the rhachitic rosary.

The new bone on the parietal eminences gives rise to a peculiar shape of the head—a squareness that is quite characteristic. The forehead is prominent. Some patients



FIG. 54.—Rickets.

are dolichocephalic, not square-headed. The occiput is usually rather broad and flat.

Harrison's groove, the concavity of the lower part of the antero-lateral chest wall, is often quite marked in rhachitic patients. While not directly caused by the disease, its formation is evidently facilitated by the softness of the ribs. The concavity of the chest is in marked contrast to the prominent belly.

The spine usually has a long rounded convexity. The

deformity may appear more or less fixed, but, if the child be laid upon his face, and if his pelvis be lifted from the table by raising his heels in the air, pressure with the palm of the hand upon the curve will obliterate it. Many of the severe cases of rotary lateral curvature are probably rhachitic in their origin. The pelvis is often narrowed and deformed. The rhachitic pelvis in the female becomes of great interest in later life to the obstetrician.

In the lower extremity the rhachitic deformity takes the form of knock knee or bow leg. In the former the deformity is usually in the femur, in the latter in the tibia. Probably the chief cause of these deformities is the inability of the bones of the extremity to bear the weight of the body without bending, but sometimes they may be quite marked in children who have never walked. The popular idea that they are caused by too early walking, is of course erroneous, for the rickety child usually walks late, and any child will always walk as soon as he can. In the active stages of the disease, and roughly up to the end of the fourth year, these crooked bones have a more or less elastic feel when an attempt is made to bend them, but after that time they become quite hard and unyielding. As long as they are soft there is some hope of correcting the deformity with braces, but when they have become eburnated, little can be expected from conservative treatment.

The diagnosis is made on the peculiar bone changes. The severe cases rarely give rise to doubt. The milder ones are often missed. The disease is very frequent in large cities, and, if one is looking for its manifestations, one will find them in a very heavy percentage of one's cases.

Scurvy has its painful swellings in the neighborhood of the joints, its more or less acute onset, and its hemorrhages. The rhachitic spine may simulate the tuberculous spine, but

it lacks rigidity, muscular spasm and pain. Other signs of rickets can be found, and these practically rule out tuberculosis, for the association of active rickets and tuberculosis is at best very rare.

In chronic hydrocephalus the shape of the head is globular rather than square, and the epiphyses are not enlarged.

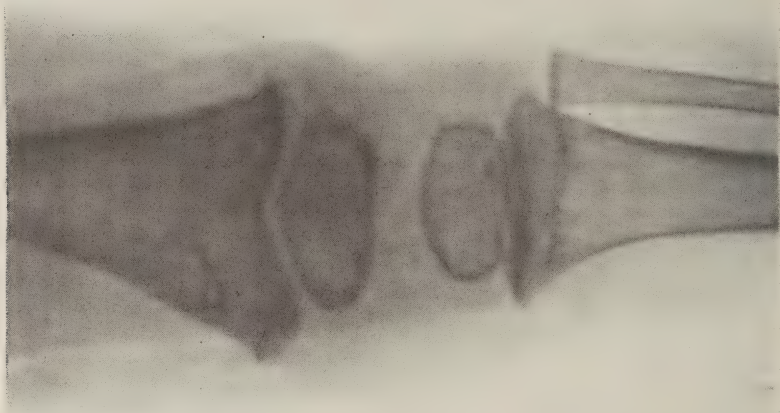


FIG. 55.—Rickets. Skiagram of a knee.

TREATMENT.—Of the first importance in the active stages are a well regulated diet and plenty of fresh air. Findlay stresses the importance of exercise, but manifestly a child with rhachitic deformities in its lower extremities, should be kept off his feet as much as possible. Otherwise, unless he wear braces his deformities will increase. Most authorities, in addition to a proper diet, recommend the administration of cod liver oil. Some of them believe also in the efficacy of phosphorus.

It is seen that the treatment of rickets as a disease, is along medical lines. The deformities which follow the disease present a different problem. If they are attacked early, before the bones become sclerosed, their treatment usually is not very difficult. The round back, or the scoliotic spine, demands recumbency, preferably on some such apparatus as the Whitman-Bradford frame.

Mild degrees of bow-leg or knock-knee may be treated by frequent manipulation. The mother is shown how to bend the limb as if it were a crooked stick which she were trying to straighten. She should manipulate it fifty or a hundred times a day. In addition the sole of the shoe is raised about a quarter of an inch, on the inner side for knock-knee, on the outer for bow-leg.

More marked cases demand brace treatment. Standard forms of braces are in use, but it is not difficult for anyone with a little mechanical ability to devise one that will answer the purpose perfectly.

Severe cases of bow-leg and knock-knee demand operative treatment, as do moderate cases after the bones have become hardened, that is, after the end of about the fourth year. Osteotomy is not a difficult operation, and if done with ordinary care is comparatively free from risk. A sharp osteotome incises the tissues longitudinally down to, and through, the periosteum, at the seat of the greatest deformity. In bow-legs this is usually at the junction of the middle and proximal thirds of the tibia, in knock-knee just proximal to the condyles of the femur. After the periosteum has been incised longitudinally, the blade of the osteotome is turned transversely and with blows of a mallet divides partially the bone. The fracture is completed by manual force. After every few strokes of the mallet the operator moves the osteotome back and forward to keep it from wedging fast.

One or two sutures are inserted in the skin, a sterile dressing is applied, and the limb is put up in plaster of Paris in a slightly overcorrected attitude. The plaster of course extends well above and below the seat of the fracture, and remains on for about two months. If the bones are still soft, braces should be applied when the plaster is removed.

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SECTION IV.

CHRONIC ARTHRITIS

CHAPTER I

THE TWO GREAT TYPES

JOINT TUBERCULOSIS IN GENERAL

THE custom is almost universal to describe the chronic arthritides as separate from inflammations in the shafts. This custom has decided merit, for, when the joint is involved, the symptoms of the arthritis usually dominate the picture, and throw the symptoms referable to the bone decidedly in the background. The fact must not be forgotten however that the distinction is largely an artificial one, that no sharp dividing line can be drawn between an arthritis and a myelitis, that most arthritides are the result of a previously existing myelitis, and that a primary arthritis may easily spread into the bone and involve the bone marrow.

The whole subject of chronic arthritis is a most confused one in all its aspects. Indeed this confusion exists not only as to its ætiology and its pathology but also as to its classification and nomenclature, so that often a student is baffled in his efforts to find out an author's meaning.

Until comparatively recently practically all these arthritides were grouped under the broad general name of chronic rheumatism, and were supposed to be due to some mysterious dyscrasia or diathesis, and even to this day we find some investigators taking refuge in the similar terms rheumatoid and metabolic. It is interesting in studying chronic arthritis to see how a firm structure of fact slowly has been built up by patient investigation. On the other hand at all times this structure of fact has been almost

buried in a flood of supposition and theory. One by one, various members of this group of diseases have been identified and described, until now it may be said that the main features of the majority of them are fairly well known, and the pathological characteristics of the others.

CLASSIFICATION.—The prevailing method of classifying the chronic arthritides is partly on an ætiological, and partly on a pathological and clinical basis. Thus, most writers describe tuberculous, gonococcic, syphilitic and other forms of arthritis, and then classify the cases of unknown ætiology according to some pathological or clinical feature which they deem most important. The disadvantages of this method are manifest. The ideal classification of course is an ætiological one, but, as long as so much doubt hangs about the cause of several forms of chronic arthritis, it is well if possible to classify on a clinical or a pathological basis, preferably the latter. As has been said, the changes, especially the gross changes observed in the tissues of the joint are comparatively few in number, and on the basis of these it is possible to divide all cases of chronic arthritis into two great divisions or types, whose pathological features are sharply differentiated and whose clinical features usually are so well marked as to permit a distinction between them. They may be differentiated almost invariably, or invariably, with the Röntgen ray. The first type is characterized by a proliferative inflammation in the synovial membrane and in the bone marrow, with a resulting rarefaction or death of the bone, and a perforation or death of the articular cartilage. This type probably includes all the bacterial arthritides, *e.g.*, tuberculous, syphilitic, pneumococcic, typhoid and coccidioidal arthritis; and the cases of chronic

arthritis supposed to be caused by diplostreptococcic infection in the tonsil, in the deep urethra, and in other locations. The English often call these last cases "rheumatoid" arthritis, Goldthwait calls them "atrophic" and "infectious" arthritis, Nichols and Richardson call them "the proliferative form," and other writers have given them other names, almost without number.

An arthritis of this type may recover completely, or it may result in fibrous or in bony ankylosis. All the cases in this type are alike, clinically and radiographically. They all belong in the same family, so to speak. While the different members of the family can be often diagnosed clinically, a positive diagnosis can be made only in the laboratory.

The second great type has been recognized as a clinical entity for a number of years, though its cause has never been established. Its gross pathological feature is the formation of new bone at the joint line, with the production of spurs and bony ridges at the lines of insertion of the capsule, but the essential original pathological change at the bottom of this bone production never has been determined until recently. This is the senile type of arthritis, the "arthritis deformans" of the Germans, the "osteoarthritis" of the English, the "hypertrophic arthritis" of Goldthwait, the "degenerative form" of Nichols and Richardson, and the "metabolic" arthritis of certain other writers. The joint becomes distorted and mechanically damaged, but union of the bones entering into it, whether by fibrous or bony tissue never takes place except in the spine. On the other hand the bony changes are permanent, and a joint once damaged by this form of arthritis probably never returns to a completely normal state.

Into one of these two great types falls every case of chronic arthritis. They can usually be distinguished clinically and always by the X-ray. While every chronically inflamed joint belongs quite definitely in one class or the other, nevertheless in rare instances a patient may show signs of the one great type in certain joints, and of the other great type in others, but it is doubtful if the involvement is ever synchronous.

THE FIRST GREAT TYPE OF CHRONIC ARTHRITIS TUBERCULOUS ARTHRITIS

ÆTIOLOGY.—The exciting cause of joint tuberculosis is, in every instance, the tubercle bacillus. Authorities differ as to the relative frequency of the bovine and human type of the organism as a causative agent. Except in the very rare instances of direct infection from the outside, practically unknown, the tubercle bacillus must be brought to the joint in the circulation.¹ The consensus of opinion is that it comes in the blood, either floating free in the blood stream or in the embrace of a leucocyte. Friedrich² maintains that the infection may be through the lymphatics. Though occasionally an infected embolus may be the starting point of the disease, a macroscopic plug of infected tissue as a causative agent is probably a great rarity. Marrow tuberculosis may be easily produced in laboratory animals by the injection of a pure culture of tubercle bacilli into the nutrient artery,^{3,4} as well as by carrying them in on a platinum loop through a trephine opening in the cortex.^{5,6}

CONTRIBUTING CAUSES.—Heredity and environment may be considered together under this heading, for they are not always easy to separate in their influence. There is a type of physique considered prone to tuberculous

¹ KAPPIS: "Beitrag zur traumatischen Tuberkulose." *Deut. med. Woch.*, 1910, xxxvi, 1310.

² FRIEDRICH: "Experimentelle Beitræge. u.s.w." *Deut. Zeit. f. Chir.* 1899, liii, 512.

³ MUELLER: "Experimentelle Erzeugung typischer Knochentuberkulose." *Cent. f. Chir.*, 1878-79, xi, 317.

⁴ HUETER: "Die experimentelle Erzeugung der Synovitis granulosa, etc." *Deut. Zeit. f. Chir.*, 1878-79, xi, 317, 330.

⁵ ELY, LEONARD W.: "Lymphoid marrow and tuberculosis; an experimental study." *J. A. M. A.*, 1915, lxxv, 1868.

⁶ OLIVER, JEAN: "Early changes following the injection of tubercle bacilli into the metaphysis of the long bones of animals." *Jour. Expr. Med.*, 1920, xxxii, 153.

infection—the so-called tuberculous diathesis—and the offspring of tuberculous parents is notoriously vulnerable to tuberculosis, but how much of this vulnerability is due to constitutional predisposition and how much to the infection spread by careless tuberculous parents is still a subject of debate.

The same uncertainty exists as to the influence of environment. The disease is rather frequent in the dense population of the larger cities, and its frequency has been ascribed to the effect upon the constitution of general insanitary conditions, but the element of infection here also is probably of much greater importance than is the lowered vitality.⁷

TRAUMA.—As in most diseases of the bones and joints, so in tuberculosis, trauma has been considered an important element in the causation. The subject has been attacked from the experimental side with contradictory results.^{8,9,10} Clinicians differ markedly in their estimation of the importance of injury. Some ascribe to it a very prominent role, others disregard it entirely. All authorities agree that a severe injury, such as a fracture or a dislocation, is practically never followed by tuberculosis of the bone or joint. The injury is a slighter one, such as a “strain” or a sprain. Indeed, as we shall see, some writers have maintained that the vulnerability of the joints to injury determines the location of tuberculosis in their vicinity. On the other

⁷ In this connection the experience of San Francisco is interesting. A marked decrease in the number of cases of joint tuberculosis following the great fire of 1906 has been noted.

⁸ KRAUSE: “Tuberkulose der Knochen und Gelenke.” Leipzig; F. C. Vogel, 1891.

⁹ FRIEDRICH: “Experimentelle Beitræge zur Kenntniss der chirurgischen Tuberkulose.” *Deut. Zeit. f. Chir.*, 1891, liii, No. 16.

¹⁰ HONSELL: “Ueber Trauma und Gelenktuberkulose.” *Beit. z. klin. Chir.*, 1900, xxviii, 659.

hand, the normal joint is built to withstand the ordinary injuries to which it is exposed, and it is hard to believe that an injury so slight as to escape the notice of the patient or of his parents, could be much of a factor. The tendency of the patient to ascribe his disease to an injury, must be borne in mind, even to an injury long antecedent. Again no trauma other than a fracture can possibly affect the bone marrow, where joint tuberculosis in the majority of cases begins. Sprengel has called attention to the fact that those joints which are most often injured, such as the joints of the hand and of the foot, are relatively infrequently affected with tuberculosis.

The most reasonable view yet advanced as to the relation of trauma to joint tuberculosis, is the following: Strictly speaking, trauma plays no role in the causation of the disease itself. It simply lights up an already existing, perhaps quiescent, disease. Examination of specimens of bones in the laboratory indicates that tuberculosis can exist, perhaps indefinitely in the bone marrow without involving the joint. Again, fairly extensive involvement of the synovial membrane may give rise to symptoms so insignificant as not to draw the patient's attention to the disease; or a tuberculosis of the synovial membrane, possibly diagnosed at the time of its occurrence as "rheumatism," or dismissed without a diagnosis, may become completely encapsulated and may be forgotten. In such a case trauma tears the fibrous adhesions loose, and sets the infectious material free in the joint, and the patient naturally dates his disease from the injury. The trauma of operation seems especially to light up joint tuberculosis, as when an inflamed joint is explored for diagnosis, or when an operation is undertaken with an erroneous diagnosis.

The sequence of joint tuberculosis on certain acute

infectious diseases has often been noted, and these diseases, somewhat in the order of their importance, are, in the child, measles, whooping cough and scarlet fever, and in the adult, pneumonia and typhoid. The consensus of opinion is that they lower the patient's vitality and so predispose to the invasion of tuberculosis, but it is far more likely that they cause an actual morphological change in the bone marrow, which renders it a more suitable pabulum for the tubercle bacillus. We know that infections cause profound changes in the bone marrow, easily recognized by the naked eye or under the microscope, changes which are much more easily appreciated than are the somewhat vague terms "lowered vitality" and "decreased resistance." Again, the diseases which most depress the vitality are not those most often followed by joint tuberculosis.

OCCURRENCE.—Joint tuberculosis occurs at all ages. It is frequent in childhood, but very rare in the first year of life, perhaps because the portion of the bone most liable to invasion is then largely composed of cartilage, whose resistance to tuberculosis is well known. Roelsing¹¹ maintains that synovial tuberculosis is fairly frequent in the first year of life.

The male sex furnishes a small majority of the cases of joint tuberculosis, not because it is more liable to trauma, but because it is more active. What effect function has we shall see later.

The liability of the various joints to invasion is roughly proportional to their size. The sacro-iliac joint, with its infrequent involvement, forms an exception to this rule. Observers differ somewhat in their statistics, but agree

¹¹ ROESLING, TH.: "Ueber tuberculoese Arthritis im fruehesten Kindesalter." *Arch. f. klin. Chir.*, 1896, liii, 620.

fairly well in the main. According to Whitman¹² the spine is most frequently affected, then the hip, then the knee, ankle and tarsus, elbow, wrist, and the shoulder. Tuberculosis of the clavicular joints is rare, and of the joints of the fingers and toes very uncommon, though tuberculosis of the shafts of the metacarpals and phalanges is fairly frequent in childhood.

PATHOLOGY.—The essentials of the disease are the same as are those of tuberculosis anywhere in the body, but its course is modified by the presence of the joint cavity and of the rigid bony shell. Only two tissues in and about the joints are vulnerable to the tubercle bacillus unassociated with any other organism, except of course in the case of a general miliary tuberculosis, and these two tissues are the bone marrow and the synovial membrane.¹³ Most authorities agree that the disease may start in either of these tissues, but Nichols¹⁴ maintains that the origin is always in the bone marrow. Basing my opinion upon analogy, upon clinical experience, and upon the examination of material in the laboratory, I hold the former view, although it must be said that Nichols has made out a strong case.

TUBERCULOSIS of the shafts of the long bones, except of the phalanges and the metacarpals is rare in this country, though it is said to be more frequent abroad.^{15,16} The disease attacks the short and the flat bones, and especially

¹² WHITMAN, ROYAL: "Orthopædic Surgery." Philadelphia and New York, Lea and Febiger, 1919, 6th ed.

¹³ ELY, LEONARD W.: "Joint Tuberculosis." New York, Wm. Wood & Co., 1911.

¹⁴ NICHOLS, E. H.: "Tuberculosis of the bones and joints." *Tr. Am. Orthop. Ass.*, Philia. 1898, xi, 353.

¹⁵ FRAZER, JOHN: "Tuberculosis of the bones and joints in children." London, A. & C. Black 1914.

¹⁶ FRIEDLANDER: "Die tuberkulöse Osteomyelitis der Diaphysen langer Röhrenknochen." *Deut. Zeit. f. Chir.*, 1904, lxxiii, 249.

and with greatest frequency, the ends of the long bones. This marked predilection of tuberculosis for the bone in the region of the joint has been the subject of much discussion, and has been explained on various hypotheses, such as the abundant blood supply in the marrow in the vicinity, the slowness of the blood stream in the spongy bone, and the liability of the bone here to injury. Lexer has advanced the ingenious theory that the arrangement of the blood vessels in the vicinity of the joint is the cause of the deposition of the tubercle bacilli. In the young, the arteries about the epiphysial disc are largely end arteries, and this fact favors the lodgment of the supposititious tuberculous embolus. Without entering here into all the details of the argument, it is sufficient to say that none of these theories furnishes a satisfactory explanation.¹⁷ The enquiry is not an idle one. Its correct answer not only is important for an understanding of the pathology and of the ætiology, but also has a bearing on all our ideas on the treatment.

Tuberculosis flourishes in the region of the joints because the tubercle bacillus finds there its appropriate food supply. While the structure of the marrow is most diverse, and while its constituents vary greatly, not only in different individuals but also in the same individual at different times; nevertheless the bone marrow is generally divided into three main classes, namely lymphoid, fatty, and "myxomatous" or fibrous. The resistance of fatty and fibrous tissues to an unmixed tuberculous infection is well known. These tissues make up the bulk of the two kinds of marrow that bear their names, and they are practically immune to a pure tuberculous invasion. Lymphoid marrow, on the other hand, like lymphoid tissue anywhere in the body, is vulnerable to the tubercle bacillus.

¹⁷ *Vide* previous writings of the author on this subject.

In the young practically all marrow is lymphoid. As age advances the lymphoid marrow gives place to fatty marrow, and this in turn generally to fibrous marrow. The change begins in the shaft of the long bones, and the lymphoid marrow persists longer in their extremities, finally disappearing there as well. This is in the human being, of course, and is not true of all other animals. In adult rabbits the marrow of the shafts, as well as that of the extremities of the long bones, is of the lymphoid variety. In the human animal, lymphoid marrow is found in the bodies of the vertebrae after it has disappeared from the extremities of the long bones. Whether it persists there into old age I cannot say of my own knowledge.

When we study the incidence of bone or rather marrow tuberculosis we find that it follows closely the distribution of the lymphoid marrow, that is, as long as it is not complicated by a secondary infection, and that where no red or lymphoid marrow is, there the bone is immune, or practically immune, to invasion by the tubercle bacillus. This rule applies only to the disease as it occurs naturally, for it has been my experience that if a pure culture of tubercle bacilli be inserted into the tissues of laboratory animals it will grow, no matter how resistant the animal may be, or how unfavorable a medium the tissue.^{18,19,20}

Lymphoid marrow in the adult generally is found in the spongy bone, and its presence in the extremities of the long bones is dependent upon function in the joint. When function is abolished in the joint, the lymphoid mar-

¹⁸ ELY, LEONARD W.: "Lymphoid marrow and tuberculosis," *Jour. Am. Med. Ass.*, 1915, lxx, 1868.

¹⁹ ELY, LEONARD W.: "Experimental tuberculosis of muscle. Bone and joint studies I." Stanford University, Cal., 1916. Published by the University.

²⁰ OLIVER, JEAN: "Early changes following the injection of the tubercle bacillus into the metaphysis of long bones." *Journ. Exper. Med.*, 1920, xxxii, 153.

row tends to disappear. According to Ollier,²¹ Mauclore,²² and Lagrange²³ after bony union has taken place, following a resection, the spongy bone in the neighborhood slowly disappears and the cortex thickens. The central marrow canals of the adjoining bones slowly lengthen and approach each other. Eventually they may join, and the canalization of the former site of the joint may become complete, but this is probably the exception. In some of my cases of resected knees I have had the chance to watch with the aid of the X-rays the rearrangement of the bone, and I have noted this tendency to canalization, but I have never seen the canalization complete.

Another important change which follows a bony ankylosis, is the complete disappearance of the synovial membrane, as has been mentioned in the general section.

The reason for the usual site of tuberculosis in the ends of the long bones, therefore, is the presence there of lymphoid marrow.

Not only is the relation of lymphoid marrow to tuberculosis important for a comprehension of the pathology of the disease as it exists in bone, but also its recognition furnishes us with a definite principle of treatment, and enables us to understand many of the aspects of bone and joint tuberculosis hitherto obscure, such as the frequency of the disease in children, its recovery after resection, its improvement under rest, etc.

The original tubercle is formed in the marrow in the vicinity of the joint. In the short and in the flat bones, it may be formed anywhere of course, though even in these bones the disease chooses certain favorite sites. In the long bones the favorite starting place in children is in the

²¹ OLLIER: "*Dict. encycl. des Sciences Médicales.*" Paris, 1870, page 191.

²² MAUCLAIRE, P.: "*Nouveau traité de Chirurgie.*" Paris, 1909, page 235.

²³ LAGRANGE: "*Traité de Chirurgie.*" Paris, 1901.

neighborhood of the epiphysial cartilage, probably on its shaft side. In the adult it probably starts nearer to the joint line itself, that is, nearer to the actual end of the bone.

The custom in the past has been rather widespread, to regard as the typical original lesion a tuberculous infarct,

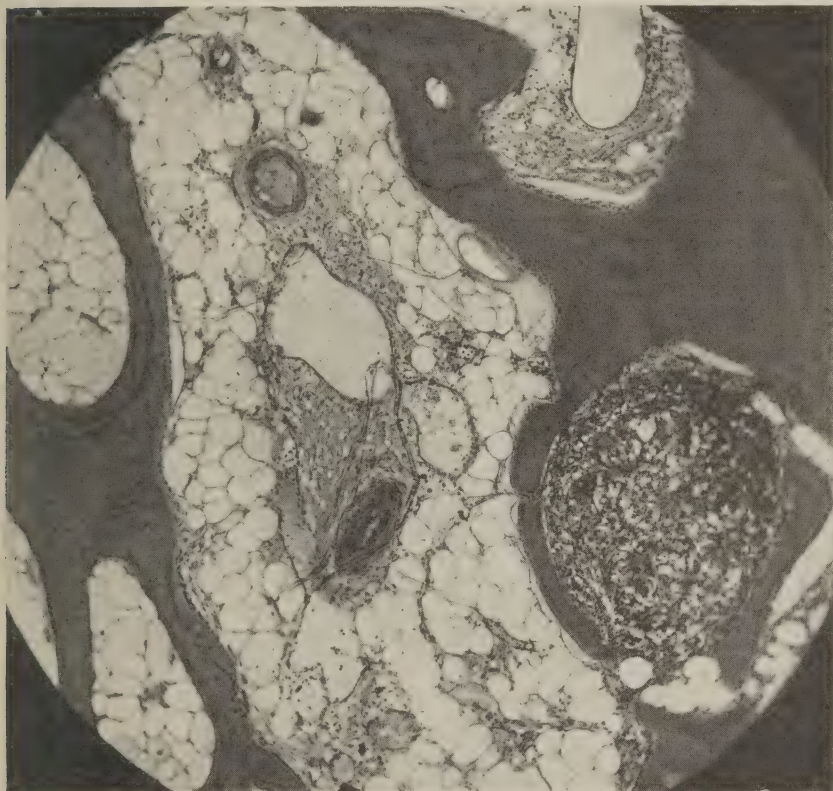


FIG. 56.—Discrete tuberculosis in the bone marrow. Low power photomicrograph.

which was supposed to give a pyramidal shape to the tuberculous process in the bone end, with its base abutting on the joint. Possibly the disease occasionally may begin in this manner, but here, as elsewhere in the body, it probably starts as a solitary tubercle caused by the deposition in the lymphoid marrow, of tubercle bacilli, which have been

brought there either in the embrace of a leucocyte, or else floating free in the blood stream. There is nothing different in tuberculosis of the marrow and of a lymph node, except that the former runs its course enclosed in a rigid shell, and usually in the neighborhood of a joint.

From the time of the formation of the original tubercle, the disease tends to spread at the periphery, with the same tendency to the formation of fresh tubercles, and to breaking down as is observed in other organs. Nature on her part tries to wall it in with fibrous tissue and, to a lesser extent, with bone, and according as one or the other of these processes prevails, the disease tends to spread or to recover. It is quite possible that many cases of marrow tuberculosis are healed by nature without ever having been recognized clinically, as are many cases of pulmonary tuberculosis. The symptoms are not marked unless the disease is in the neighborhood of a joint. In fact, we know well that when bony union follows the resection of a tuberculous joint, provided only that a pus infection be not added, even large amounts of tuberculous tissue left behind at the operation are without significance, and may remain locked up in the bone indefinitely, causing no symptoms unless they are brought back into activity by operative measures.

Starting in the end of the bone the disease may spread in any direction, and generally as far as the lymphoid marrow extends, though of course it must be said that authorities still differ as to the role of the lymphoid marrow in the process. It may extend shaftward, or outward toward the periosteum. In the latter case the tuberculous granulations may break through the thin cortex and the periosteum, and eventually gain the surface without joint involvement. This is rare, very rare. Tuberculosis seems

to seek the joint as if with a well-defined purpose, taking such a path and affecting the bone and the cartilage in such a way, and causing such clinical symptoms that one

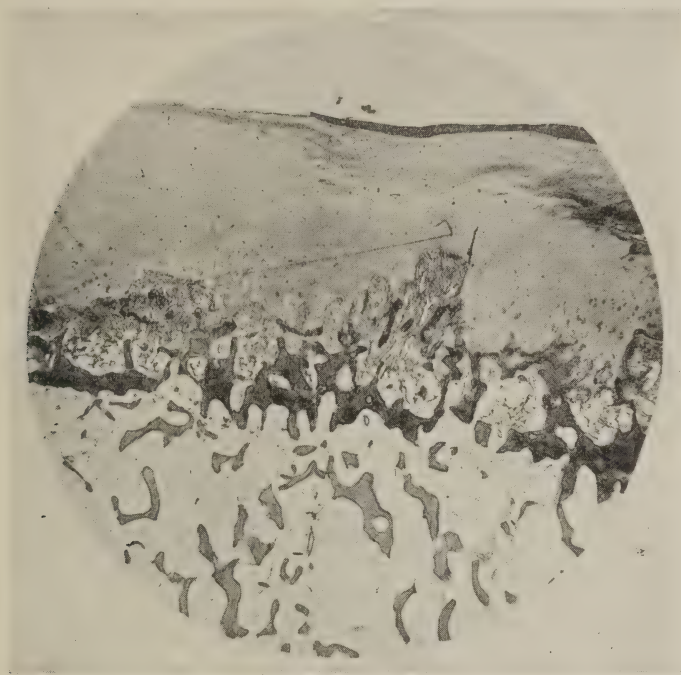


FIG. 57.—Tuberculosis in the bone marrow immediately beneath the articular cartilage, low power photomicrograph. Note the degeneration of the cartilage at its surface, the "fingers" of tuberculous granulations pushing up into it, the osteoid tissue beneath this, and the rarefied bone at the bottom.

can make a *fairly* reliable diagnosis of the disease by its effect upon the bone as revealed by the Röntgen rays.

The tuberculous granulations may reach the joint by breaking through at the margin of the cartilage where it joins the ligament; or, spreading along under the cartilage, they may destroy its nutrition in spots, kill the cartilage, and push up through it in the shape of little fingers or columns; or, they may spread under wide areas of it, and, killing it in whole or in part, cast it off a loose body in the

joint. When the infection has burst into the joint cavity the synovial membrane becomes affected, and a tuberculous arthritis is added to the tuberculous myelitis.

The next step is the invasion of the other bone or the other bones of the articulation. Normal cartilage, carti-

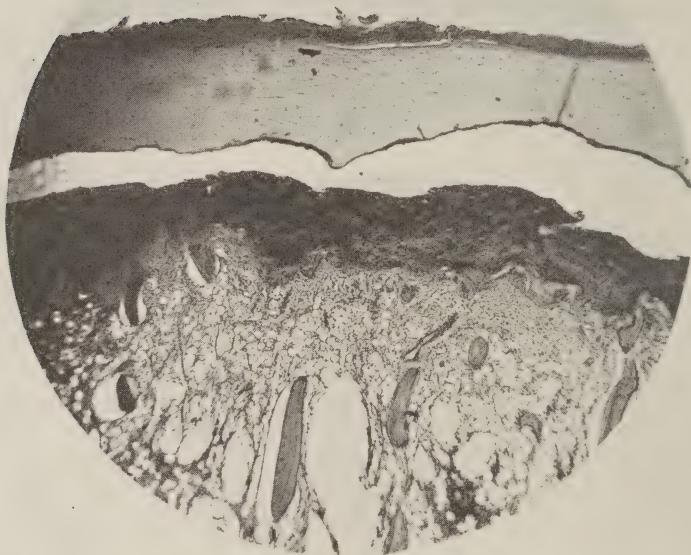


FIG. 58.—Joint tuberculosis, low power photomicrograph. The tuberculous granulations in the marrow have run along beneath the cartilage and dissected it off. Note the almost complete absence of bone immediately beneath the cartilage, and the degenerated shaggy appearance of the surface of the cartilage.

lage whose nutrition has not been disturbed, is an absolute bar against the march of tuberculosis. The articular cartilage draws its nutrition from the marrow beneath it, probably to a small extent from the vessels at its circumference, and perhaps also from the joint fluid. It is there-

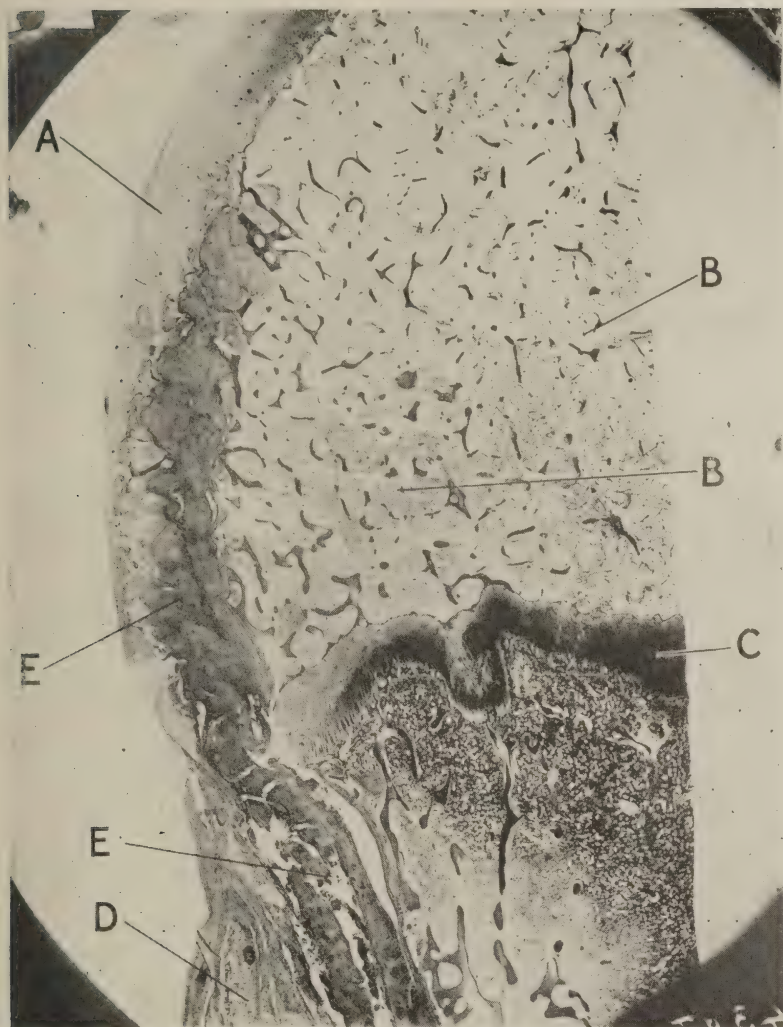


FIG. 59.—Tuberculosis of the knee-joint in a child, with secondary infection. Low power photomicrograph of the distal end of the femur. The picture shows well how the tuberculous granulations, E, E, in the metaphysis make their way at the margin of the epiphyseal disc, C, into the epiphysis, B, and run along under the articular cartilage, A, killing it and perforating it to gain access to the joint. D, periosteum.

fore practically immune to invasion from the joint side. The disease must make its way into the bone at the circum-

ference of the cartilage, where it is joined by the tuberculous synovial membrane. A process similar to that in the marrow of the other bone is then enacted here.

At any time in the course of the disease a tuberculous or cold abscess may form in the bone or in the joint, rupture the capsule, gain the surface and become infected. As we have seen, simple uncomplicated tuberculosis is a disease of lymphoid marrow and of the synovial membrane, following certain paths, and never involving other tissues. With a secondary infection the whole typical picture changes, and the mixed infection may run riot through all the tissues in and about the joint.

When the disease starts in the synovial membrane it may stay indefinitely in this structure, involving the entire membrane or only a part of it according to circumstances, or at any time it may invade one or more bones of the articulation, spreading under the margin of the joint cartilage where it fuses with the synovial membrane, to gain access to the marrow. From then on the progress of the disease is the same as in a case with a so-called bone focus.

We see then that it is perfectly possible to have a tuberculous osteomyelitis without an arthritis or synovitis, and probably also a tuberculous synovitis or arthritis without an osteomyelitis.

MARROW CHANGES.—The favorite starting point of the disease in the child probably is not, as is often said, in the epiphysis, but just to the shaft side of the epiphysial disc. In the adult, the primary focus may be anywhere in the end of the bone, but other things being equal, it chooses a site where the bone is least dense. We speak very positively of the site of the original focus, but of course it is largely a matter of surmise, based upon the X-ray findings

and upon the examination of the material removed at operation. Often some part of the bone will show an involvement out of all proportion to that of the rest, and we assume this to be the original focus.

There is nothing characteristic about the shape of the tuberculous process. It may be wedge shape, with the base toward the joint, but in the vast majority of cases it is not. If it has a characteristic in this respect, it is irregularity. The tubercles may be discrete, and scattered widely through the marrow, or larger and smaller portions of the marrow may be nothing but masses of tuberculous granulations. Here, as elsewhere in the body, some cases show a marked tendency to rapid spread, with diffuse invasion, and necrosis and breaking down—cheesy degeneration—while the slower, more benign cases are characterized by fibrous encapsulation and discrete tubercles. Fibrosis of the marrow may be regarded as nature's protective reaction. It often reaches a considerable degree. A fatty change is especially prominent in the old cases complicated by a secondary pus infection—the so-called fatty osteomalacia. In old cases calcification of the necrotic material is often a prominent feature, and this calcified material is responsible for the shadows seen in the X-ray plate—shadows sometimes mistaken for new bone. New bone formation is never a feature of joint tuberculosis, though occasionally about old, healed, or almost healed foci, new bone, never enough to show in the X-ray plate, may be seen in the laboratory specimen.

The blood vessels of the marrow often show thickened walls. The engorgement, so prominent in acute infections, is not seen. More or less peculiar to tuberculosis is the band of osteomyelitis immediately under the joint cartilages. The results of this upon the bone and cartilage

often cause a picture which enables one to diagnose the disease fairly accurately with the Röntgen rays.

Healthy marrow may of course be found in the midst of diseased. Hence we are not justified in ruling out

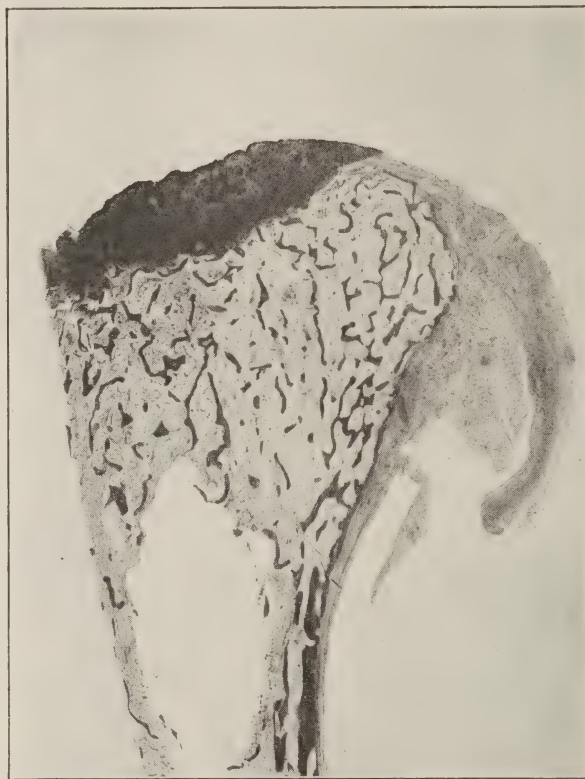


FIG. 60.—Tuberculosis of the metacarpo-phalangeal joint, with secondary infection. Note the absence of the cartilage, the marked involvement of the bone marrow at the articular surface, and the long streamers in the synovial membrane.

tuberculosis from an examination of one small piece of bone in the laboratory. Often an extended search is necessary.

BONE CHANGES.—All changes in the bone are to be regarded as passive, the result of changes in the marrow. The characteristic change is a rarefaction. The bone

trabeculae become thinner and scantier, and more permeable to the X-rays. In the milder cases, especially if they have had efficient treatment, this may be the only change observed. If the disease be more severe the bone is killed. It dies in small pieces—the so-called bone sand—or in large pieces—sequestra. A sequestrum consists of dead bone and dead marrow, may be of any size or shape, and sometimes is found fairly well encapsulated by the surrounding bone. It may remain indefinitely at the site of its formation, and need not be cast off like the sequestrum of a suppurative osteomyelitis, though it is doubtful if blood vessels ever can push their way into it, absorb it, and replace it with new bone.

In old cases, especially in those complicated by a pus infection, the bone may become so thin and soft that it can be cut with a knife. Contrary to what one would expect in such circumstances fracture is extremely rare in tuberculous bones, so rare that its presence in a suspected case speaks strongly against the diagnosis of tuberculosis.

A tuberculous focus in the end of a young child's bone sometimes stimulates the growth of bone in the neighboring epiphysial disc, but this stimulation is never great, and is only temporary. Thereafter growth lags behind, and when full stature has been attained, the affected limb is almost invariably shorter than its fellow. The resulting shortening may be due to one or all of three things, *viz.*, to subluxation, especially of the hip or knee, to damage to the bone in the epiphysis or to the epiphysial cartilage, and to the atrophy of disuse.

Rarefaction is not confined to the bone in the region of the affected joint, but is observed also in all the bones of the extremity. This is the atrophy of disuse. In this atrophy

all the tissues of the extremity share. The muscular shrinking is especially noticeable, and accentuates the appearance of the swelling in the joint.

Under the microscope the bone is seen to be less dense than normal, and the trabeculæ to be scantier and more slender. Typical rarefying osteitis, with its classical so-called osteoclasts in Howship's lacunæ, is not as frequent as one would expect. Instead the trabeculæ are often edged by cells with the appearance of osteoblasts. Sometimes these cells are separated from the trabeculæ by a translucent zone, presumably bone undergoing absorption.

As a rule in the more benign cases, bone absorption exceeds bone necrosis. It prevails especially in the zone immediately under the joint cartilage. The bony buttress disappears, and the cartilage lies upon an irregular band of tuberculous granulation tissue, containing a few small remnants of bone trabeculæ.

THE JOINT CARTILAGE.—The cartilage is roughened, irregular and fibrous. It may be dead in whole or in part. It may lie on the bone end, scarcely attached here and there or it may be loose in the joint. Parts of it, though badly damaged, may still remain, while other parts have been replaced by tuberculous granulations springing from the marrow beneath. Areas of bare bone are not seen, as in the second great type of chronic arthritis.

These cartilage changes are so constant in tuberculous joints that they are regarded as characteristic of the disease, but they are shared by all the members of the first great type of chronic arthritis, though they are rarely present in as pronounced a degree. On the other hand they may be absent altogether. Occasionally one opens a tuberculous joint at operation, and finds the cartilage smooth, glistening, and apparently normal. Sometimes the pres-

ence of a focus in the bone will be betrayed by a small dimple or irregularity in the overlying cartilage.

The joint cartilage is lessened in area by the encroach-

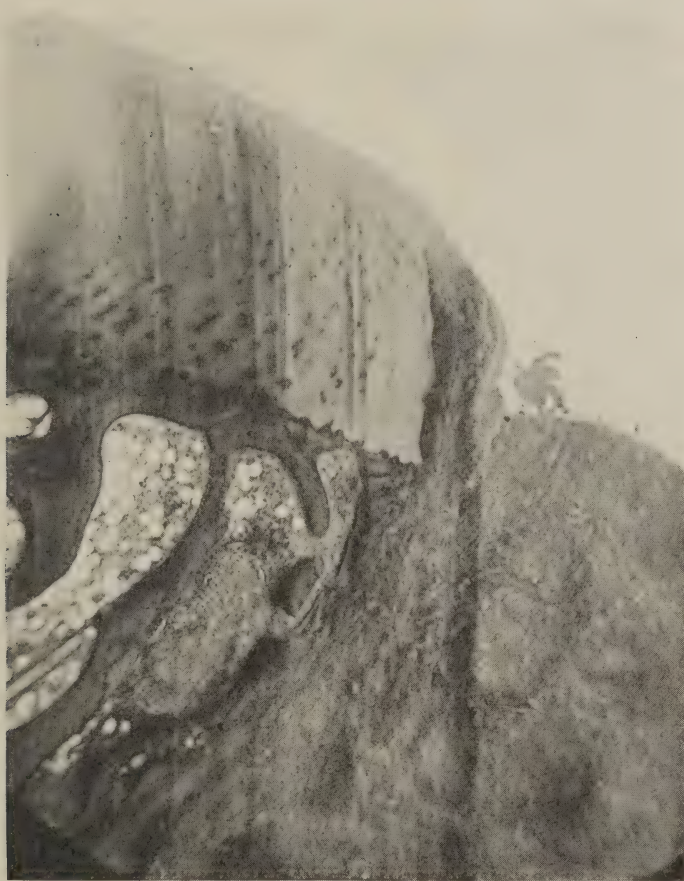


FIG. 61.—Photomicrograph, low power, from a case of tuberculous arthritis. Cartilage above and to the left, synovial membrane below to the right. Note the excellent condition of the cartilage, and the extensive involvement of the synovial membrane. Apparently the disease is just beginning to make its way into the bone at the circumference of the cartilage.

ment of the synovial membrane at its periphery. Its surface has a fibrous appearance, especially near its margin, and may be vascularized—the so-called tuberculous pan-

nus. This fibrous condition of the cartilage, this pannus, was formerly regarded as the result of the organization of layers of fibrin precipitated from the joint fluid, but the influence of any fibrin deposited upon the cartilage is probably negligible. The fibrillation is usually most prominent at and near the surface of the cartilage, and is the result of three things, *viz.*, absence of function, disease in the subjacent bone marrow, and, at the periphery, substitution of the cartilage by the synovial membrane. Under the microscope the fibrillation often can be plainly seen. As a rule, the cartilage is also thinned, and this decrease in thickness is evident in the X-ray film as an approximation of the articulating bones.

THE SYNOVIAL MEMBRANE is often thickened, succulent and inflamed. At its surface it undergoes a villous proliferation, and the villi may increase enormously in number and size, sometimes branching like moss on a rock. The resulting condition is sometimes known as lipoma arborescens. In old, slow cases the synovial membrane may consist of little else than fibrous tissue. It is seen, therefore that tuberculosis is a typical villous arthritis.

Histologically the membrane presents the typical appearance of tuberculosis, sometimes in the shape of discrete tubercles, sometimes as diffuse tuberculous infiltration. A fair guess as to the clinical course of the case can be made from an examination of the stained slide in the laboratory. The slow, more benign cases as a rule show a marked tendency to discrete tubercles, with encapsulation by fibrous tissue, while the more rapid, severe cases show diffuse infiltration, with caseation, and with little effort at fibrous encapsulation. It is this difference in nature's reaction that is responsible for the various elaborate classifications of tuberculosis. They are of doubtful value.

The borders of the synovial membrane extend at the expense of the articular cartilage especially at the surface, and in addition the enormously hypertrophied membrane may almost conceal the cartilage, but, contrary to general teaching, adhesions between the two are not a prominent feature of the disease. On the other hand, when the carti-

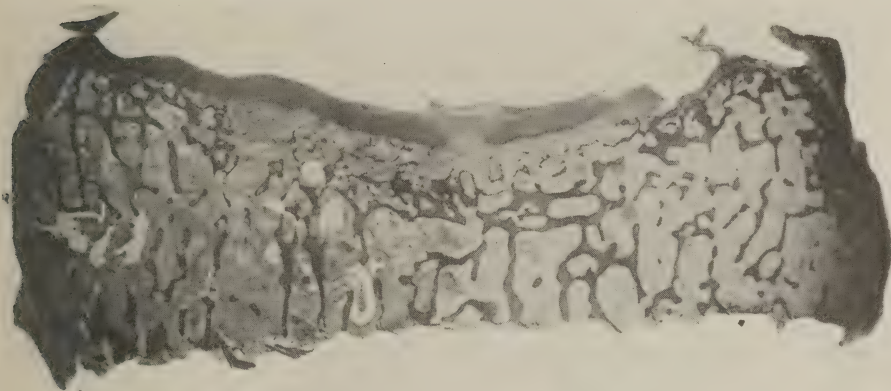


FIG. 62.—Tuberculosis of the head of the radius; photograph of the stained slide, \times about 6 diameters. Note how the tuberculous granulations in the marrow have sprouted through the articular cartilage at about its middle, and how the articular cartilage has been destroyed at the right.

lage has been thrown off, dense adhesions may form between the synovial membrane and the bone marrow.

The joint usually contains fluid. In the early stages of a marrow focus, before the disease actually has communicated with the joint, aspiration may perhaps reveal the fact that the fluid is sterile.²⁴ This is the so-called sympathetic synovitis, and is very rare if it ever occurs. The fluid usually contains tubercle bacilli, not in great numbers perhaps, and often found with difficulty by immediate examination, but capable of producing the characteristic lesions, when injected into the abdomen of the guinea-pig.

²⁴ This statement is made on the authority of others. I am not sure that I have ever seen such a case.

The fluid in a tuberculous joint may be serous, hemorrhagic, turbid or flocculent. Sometimes it may resemble pus, and it may contain detritus of bone and of cartilage. In other words, the joint may constitute a tuberculous or cold abscess. When a secondary infection is added, the joint is an abscess cavity. In the absence of a pus infec-

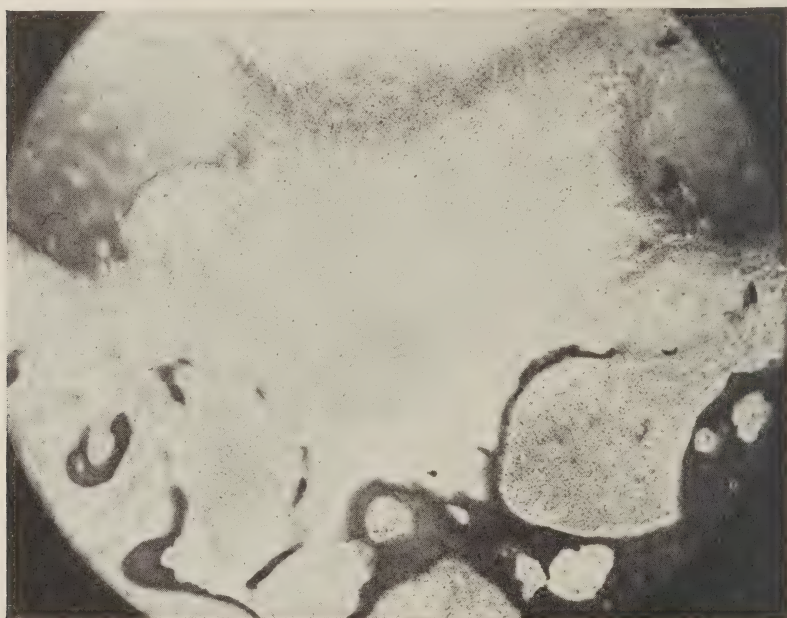


FIG. 63.—Low power photomicrograph of the central gap shown in the preceding figure. The marrow, the bone and the cartilage are dead.

tion, the fluid may be absorbed by nature, but in the presence of a secondary infection, this of course is impossible.

In the slow, dry cases, no fluid can be demonstrated in the joint, but the joint cavity is replaced by a dense mass of fibrous adhesions, which bind one bone of the articulation to the other, and both to the fibrous capsule. These fibrous adhesions, this scar tissue, are the result of the proliferative inflammation in the bone marrow and in the synovial membrane, and represent nature's effort at cure by destroying

the joint, but the effort is rarely successful in the absence of a secondary infection. Bony ankylosis seldom, if ever, results from a purely tuberculous infection.

A careful search through the mass of dense fibrous tissue will often reveal encapsulated collections of tuber-

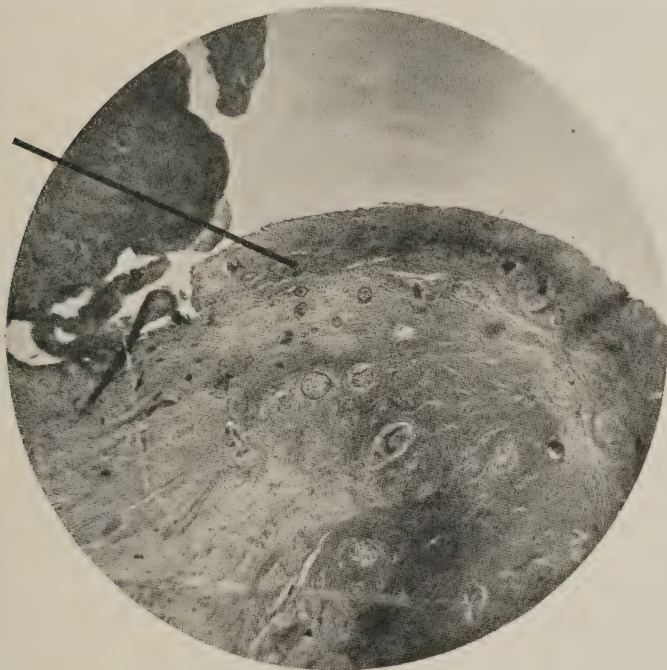


FIG. 64.—Synovial tuberculosis, low power photomicrograph. The slow benign form, with discrete tubercles and encapsulation. The general course of the disease could be guessed with reasonable accuracy from the appearance of the stained slide from which this was taken.

culous and cheesy material, capable of lighting up afresh an apparently cured disease if trauma or ill-timed operation set them free. Indeed I have seen the removal of cheesy material from the soft tissues in the vicinity of a knee which I had resected for tuberculosis ten years previously, and in which firm bony union, with prompt recovery from the disease in the bone had resulted.

The slow, dry cases with dense fibrous adhesions, some-

times are spoken of as "caries sicca," the cases with profuse production of soft granulations in the synovial membrane,



FIG. 65.—Low power photomicrograph of synovial tuberculosis, of the rapid destructive form. There is here no encapsulation, but diffuse infiltration, with cheesy degeneration—A.
B, giant cell.

as "joint fungus." Certain of the latter type of cases, especially in the knee and in the elbow, are characterized

by marked swelling. The superficial tendon sheaths are said to be involved. The muscles in the limb above and below the affected joint atrophy greatly, the skin over the joint becomes blanched, and the superficial veins prominent. This is the so-called tumor albus or white swelling.

Certain writers distinguish various pathological classes into which they divide tuberculosis in bone. There seems little more practical or scientific reason for this in disease of the bone than in disease of the lung or lymph node. Thus, the "infiltrating tuberculous lesion" is probably one in which nature's reaction of encapsulation is feeble, the "encysted tuberculous lesion" one in which it is strong. The whole form that the lesion takes is simply the result of the balance between the virulence of the infection and nature's power to resist it.

Rice Bodies are small, smooth, whitish, glistening, slippery bodies, like rice kernels or melon seeds, found sometimes in the milder forms of synovial tuberculosis of joints and of tendon sheaths. They may be few in number or many. Occasionally a collection of them is found in a capsule, but usually they are loose in the joint. They consist of fibrin and connective tissue, and may be attached by a pedicle to the capsule of the joint.^{25, 26, 27, 28, 29, 30.}

²⁵ GOLDMANN, E. E., "Ueber die Bildungsweise der Reiskörperchen, etc." *Beit. z. klin. Chir.*, 895-96, xv, 757.

²⁶ GOLDMANN, E. E.: "Ueber das reiskoerperchenhaltige Hygrom der Sehnenscheiden." *Ziegler's Beitræge*, 1890, vii, 299.

²⁷ GARRE, C.: "Die primære tuberkuloese Sehnenscheidentzündung." *Beit. z. klin. Chir.*, 1890-91, vii, 293.

²⁸ LANDOW, M.: "Ueber die Bedeutung des Faserstoffs und seine Umwandlung beim chronischen, insbesondere tuberkuloesen Hydrops fibrinosus." *Archiv. f. k. Chir.*, 1893-94, xlvii, 376.

²⁹ RIESE, H.: "Die Reiskoerperchen in tuberkuloes erkrankten Synovialsæcken." *Deut. Zeit. f. Chir.*, 1895, xlii, 1.

³⁰ KOENIG, BEDEUTUNG: "Bedeutung des Faserstoffs fuer die pathol-anat. und klin. Entwicklung der Sehnenscheidentuberkulose." *Cent. f. Chir.*, 1886, xiii, 425.

A *tuberculous or cold abscess* is a collection of broken down, necrotic material to which have been added serum and leucocytes. But for the presence of tubercle bacilli it is sterile. It may be formed in the bone and break into the joint, or it may form in the joint itself. It slowly



FIG. 66.—Low power photomicrograph of an encapsulated old cheesy collection dug out from the fibrous adhesions in an ankle joint after a resection of the talus. The patient gave a history of treatment for tuberculosis many years before, supposed to have been cured by conservative treatment. The clinical diagnosis was tuberculosis, the laboratory report read "chronic arthritis, no tuberculosis." The finding of the tubercle clinched the diagnosis.

increases in size, unlike a true abscess causing no constitutional reaction, ruptures the capsule, and makes its way into the surrounding tissues. It may then gradually disappear completely, or it may make its way slowly toward the surface, its course determined by gravity and by the path of least resistance. At any time, but especially as it nears the surface, it may be infected secondarily by

pus producing organisms. It then ceases, of course, to be a cold abscess.

The wall of a cold abscess is composed of the necrotic tissues in which it lies. When the abscess is secondarily infected it contains tubercles.

When an uninfected cold abscess ruptures spontaneously, or is opened with a knife, it discharges its contents, and its soft walls fall together. Almost invariably then, after a few days, and even in spite of the greatest care, secondary infection takes place, and the whole picture changes. To the accompaniment of a severe constitutional reaction, high fever, etc., a profuse, foul discharge starts up, and the abscess walls become thickened, infiltrated and "porky." The original focus deep in the bone is then in direct communication with the surface through a longer or shorter sinus, with the secondary infection running throughout its entire length. The condition now present is that of an infected cavity deep in the bone, whose walls cannot close in. Suppuration may go on indefinitely, and at the last may cause the death of the patient, with its complications of amyloid degeneration, tuberculosis of the viscera, or exhaustion. Meningitis, pulmonary tuberculosis, etc., are much more frequent in the presence of a secondary infection, than otherwise.

The role of the ligament and of the periosteum is not an important one in joint tuberculosis. Both play a passive part. The ligament, especially in cases treated with traction, may become loose and render the joint rather unstable. Again, in cases with much deformity, especially in the knee, the ligament may become shortened, and make the reduction of the deformity difficult or impossible.

SYMPTOMATOLOGY.—Pain is an early and constant symptom of joint tuberculosis, and is usually the cause

of the patient's call for medical help, though occasionally cases are seen in an advanced stage, that have been practically painless. The pain is more severe in children as a rule than in adults and with bone disease than with synovial. It is wont to be sharp, and sometimes comes on in severe



FIG. 67.—Old calcified tubercle in the bone marrow. It is this calcification in old cases of marrow tuberculosis that is responsible for the shadows in the x-ray picture sometimes erroneously considered as new bone formation.
Low power photomicrograph.

paroxysms. It may be felt at the seat of the disease or it may be referred. Generally, use makes it worse, but on the other hand there is a certain stiffness and pain, almost characteristic of tuberculosis, which is experienced in the morning for a while after the patient rises, and then disappears when the joint is used. This phenomenon is peculiar to the early stages of the disease.

The pain may be worse at night, coming on when the muscular spasm relaxes which has held the joint quiet. The patient cries out in his sleep, perhaps awakes, and then falls asleep again—the so-called night cries, or night terrors. The pain may intermit or it may be constant.

Sensitiveness to pressure often can be elicited if the joint is near the surface and if the synovial membrane can be reached by the finger. An inflamed synovial membrane almost always is sensitive to pressure.

Swelling and change of contour also are early and prominent signs if the joint lie near the surface, but if it be deeply located, as in the case of the hip or the spine, they will be evident only in the later stages, if at all. The swelling is caused by the fluid in the joint, by the thickening of the capsule, and by the infiltration in the surrounding tissues, never by bony thickening. The swelling tends to assume the classical fusiform shape in time, and is accentuated by the atrophy of the muscles above and below the joint. According as the amount of fluid or of granulation tissue predominate, the swelling will be fluctuating or boggy. It may be increased by abscess formation. Especially in disease of the shoulder joint, less often in disease of the knee, swelling may be absent.

Coincident with the pain, or appearing shortly after or before it, the function of the member becomes impaired. If the disease be in the lower extremity, the impairment will manifest itself by a limp, if in the upper, the arm or hand will suffer disability, if in the spine, the patient will be awkward, and will have difficulty in moving about, turning his head, bending, etc. When the joints of the spine or of the lower extremity are affected in very young children who have walked for only a short time, the patients may stop walking.

Limitation of motion is perhaps the earliest and most important physical sign. This is of course what one would expect. Disturbance of function is perhaps the first sign of inflammation in any organ, and motion is the function of a joint. The limitation is caused by pain, by muscular spasm, and by the mechanical obstruction of the inflamma-

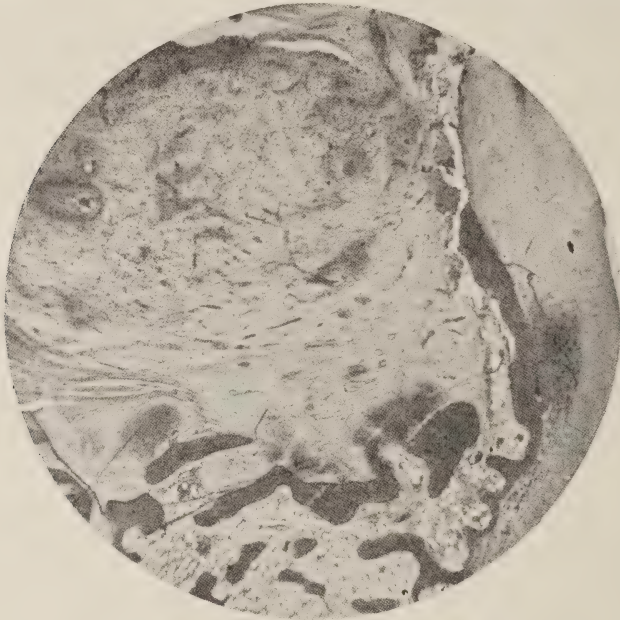


FIG. 68.—Old, well encapsulated tuberculous focus found immediately beneath the joint cartilage. At the right is the joint cartilage. Below is apparently normal bone marrow, above this, thickened bone trabeculae, above this, fibrous tissue and at the top the old tuberculous focus. The presence of this focus was indicated by a dimple in an apparently normal cartilage on the condyle of the femur from a resected knee.

tory changes in the joint structures. At first merely a restriction at extremes, it later increases, and finally may amount to a practically complete ankylosis. When the limitation is caused by muscular spasm, it disappears under a general anæsthetic; when caused by anatomical changes, it remains even under complete narcosis unless force be used to overcome it.

Muscular spasm is a most significant physical sign. It is one of the first to appear, and is almost constant. Its disappearance usually means the subsidence of activity of the inflammatory process. It represents nature's effort to hold the joint at rest, and is responsible for much of the stiffness and pain, and also for much of the impairment of function and the deformity. Formerly regarded as a pernicious symptom to be actively combated, like the pain and the disturbance of function the muscular spasm should be viewed rather as the evidence of nature's attempt at cure—as a conservative process. Muscular spasm is more pronounced in the bony type of the disease than in the synovial.

Early atrophy of the muscles moving the joint is fairly characteristic of joint tuberculosis, but not of the pure synovial disease. It is doubtless partly an atrophy of disuse, but this hypothesis does not explain the excessive muscular atrophy in tuberculosis as compared with other forms of arthritis. In the late stages of the disease the atrophy of an entire extremity may attain great proportions, especially when the case is treated by methods which abolish function in the affected extremity, but on the other hand, in hip disease treated by the method of weight bearing and the short spica, one sometimes sees an actual increase in the calf on the affected side—probably on account of the vicarious work it is called on to do.

Deformity may be said to be the sum of the muscular spasm, the swelling, the change of contour, the atrophy, and the bone destruction. In the early stages it may be slight, and only to be distinguished by careful inspection and by measurement. Each joint has peculiarities in the matter of deformity, but the general attitude of a tuberculous joint is semi-flexion. As time goes on the deformity

increases, and the flexion becomes more extreme, until at length subluxation results. Nature seems to be endeavoring to dislocate the joint, but in point of fact, she seldom, if ever, succeeds. At first the deformity can be easily corrected, especially with the aid of an anæsthetic, but later

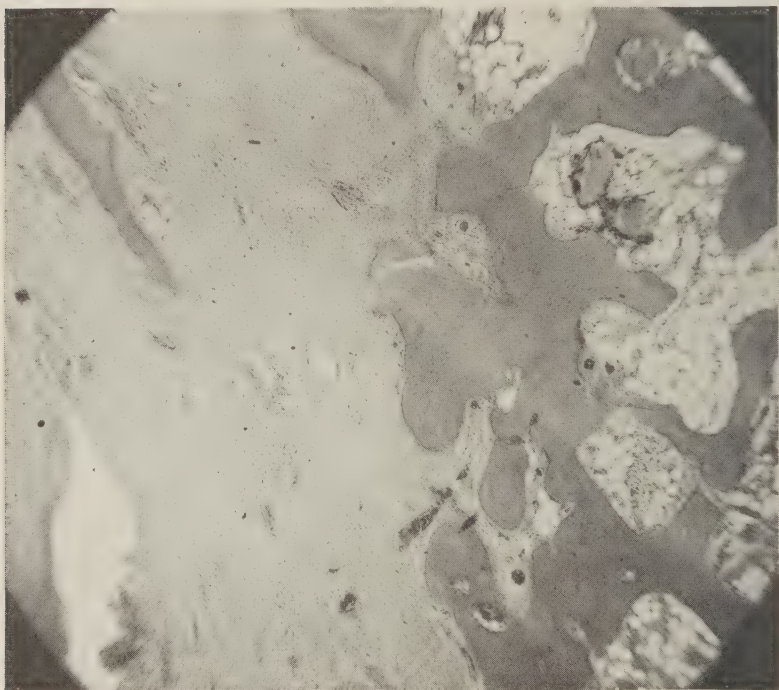


FIG. 69.—Higher power photomicrograph of a portion of the wall of the old tuberculous focus shown in the preceding illustration. This shows nature's method of walling in the disease. At the right is normal bone and marrow. Next to this come thickened bone trabeculae and then dense fibrous tissue.

the ligaments become accommodatively shortened, and the muscles also in their contracted attitude, and a firm contracture ensues, which can only be corrected with the aid of the knife, or by prolonged stretching.

A tuberculous or cold abscess may be regarded as a complication, or as a physical sign of the disease. Present

in a large proportion of cases, it is essentially a phenomenon of the late stage, and yet it may be the first thing in rare instances to draw the patient's attention to his malady, and to point the medical man to the diagnosis. It is painless and causes symptoms only by pressure upon the



FIG. 70.—Low power photomicrograph of a portion of a collection of rice bodies found incapsulated in the synovial membrane from a resected knee. The capsule shows above.

surrounding structures. Without any effect upon the constitution, and, when uninfected, by itself not influencing the course of the disease in the slightest, yet its presence bespeaks a severe form of the disease, or a feeble resistance of the patient, carries a threat of secondary infection, and immediately puts the outcome in doubt.

A cold abscess in a superficial joint like the knee or

the ankle is not difficult to recognize, but one that has broken its way through the anterior common ligament of the thoracic or lumbar spine, and has made its way along the fascial planes into the thigh, may occasion a wrong diagnosis unless one is on one's guard. It appears as an

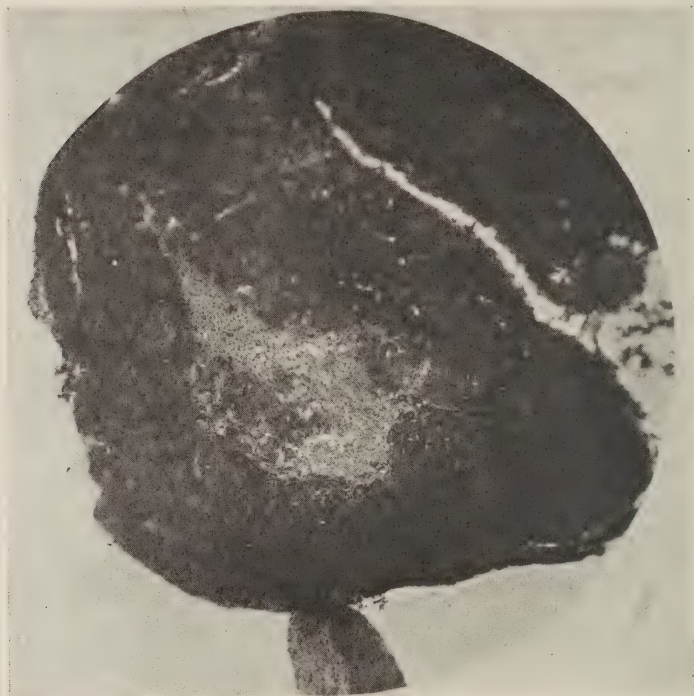


FIG. 71.—Photomicrograph of one of the rice bodies shown in the preceding figure, rather high power.

elastic, fluctuating, painless swelling, at first deeply situated, later gradually approaching the surface, and is diagnosed by establishing the presence of a tuberculous focus in the spine and then by aspiration. A cold abscess from broken-down retroperitoneal glands is diagnosed by excluding spinal disease, and then by aspiration.

A cold abscess which has ruptured spontaneously, or

has been opened with the knife, and left open, almost invariably becomes infected by pus germs, and takes a very long time to heal. Sometimes it runs for years. It communicates with the surface by sinuses whose mouths present a pale, puffy, unhealthy appearance, and which have a tendency to burrow in every direction.

Tuberculosis of a joint, uncomplicated, does not affect the general health. Often the patient has the typical appearance that makes one suspect tuberculosis, but perhaps just as often he appears perfectly strong and vigorous, especially if he be an adult. Patients with simple joint tuberculosis in favorable surroundings are often plump and well throughout the whole course of the disease. Fever is not a symptom of the disease. Its presence means the advent of a secondary infection, or of some complication such as meningitis or pulmonary tuberculosis.

DIAGNOSIS.—A good working diagnosis can be made with a reasonable degree of certainty on carefully weighed clinical evidence, but one must never forget that certainty is not to be secured without the demonstration of the tubercle bacillus. Chronic inflammation of a single joint, slowly growing worse, without evidence of venereal infection or fracture is probably tuberculous, especially in a child.

Röntgen rays show an irregular rarefaction of the bones in the vicinity of the joint, with thinning of the cartilage, and sometimes irregularity of the joint surface. In a young child the process starts usually on the shaft side of the epiphysial cartilage. In addition a carefully taken plate may show thickening of the capsule.

The ordinary tuberculin tests are not of great value, but a positive focal reaction with old tuberculin is more reliable. The patient is put to bed, and his temperature is taken every two hours for twenty-four hours. An intra-

muscular injection of old tuberculin is then given, 0.5 to 1 milligram according to age, and for the next forty-eight hours the patient's condition is watched, and his temperature is taken every two hours. A positive reaction is shown by a rise of temperature of 2 or 3 degrees Fahr., with an increase in the local joint symptoms and a greater or less general constitutional reaction.

Certainty can usually be secured by the guinea-pig test. If the joint contain fluid, it may be aspirated, and a cubic centimetre or so may be injected into the abdominal cavity of the guinea-pig. If no fluid be obtainable, the joint may be opened under strict asepsis, and a piece of the synovial membrane may be removed, emulsified with a sterile normal salt solution, and some of this may be used for the injection. The wound should be sutured immediately. The guinea-pig is killed in five or six weeks and his viscera are examined for tuberculosis. A positive guinea-pig test is final. A negative test makes tuberculosis improbable in the highest degree, so improbable that its presence may be discounted.

DIFFERENTIAL DIAGNOSIS.—The chief difficulty lies in the differentiation of tuberculosis from the other members of the first great type of chronic arthritis.

Syphilis has its history of initial lesion in the adult, and of parental infection in the child, and usually has a positive Wassermann and a negative tuberculin reaction. The patient may show other signs of syphilis. More than one joint is often involved, new periosteal bone may be discovered here and there on the shafts, and the skiagram of the joint may show certain peculiarities. Most of the damage in the child is in the epiphysis itself rather than in the metaphysis, and the epiphysial line may look furred. Sometimes in syphilis there is a marked disproportion between

the amount of damage to the bone, as revealed by the X-rays, and the joint symptoms. Indeed the bone near the joint may show great rarefaction, and the joint function may be almost perfect. This, when present, practically rules out tuberculosis. Sometimes syphilitic joints are

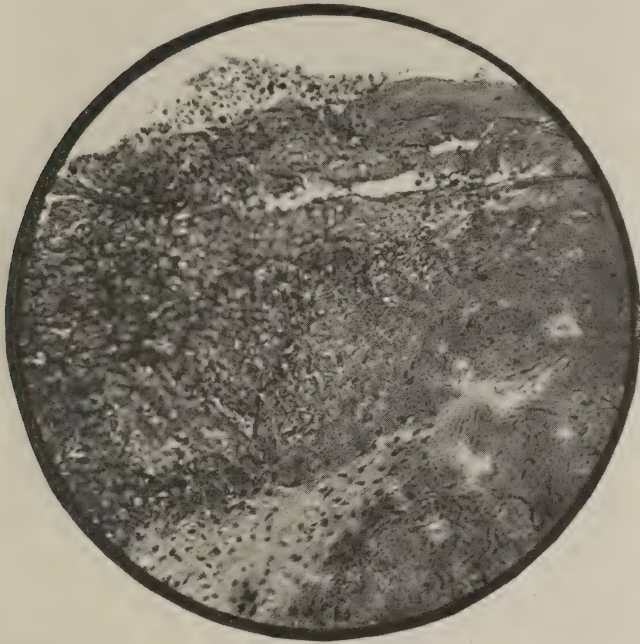


FIG. 72.—High power photomicrograph of a portion of the rice body shown in the preceding figure.

painless, but not always. Immobilization usually causes a marked subsidence of the symptoms in tuberculosis. In syphilis it is usually without effect, or may increase the discomfort. The most reliable test for syphilis is still probably the therapeutic test. Occasionally mercury and the iodides will clear up an arthritis supposed to be tuberculous.

The painful arthritides in young infants, with involvement of the bone in the neighborhood of the joints are never tuberculous. They are usually syphilitic or scorbutic.

Scurvy gives an acute swelling in the region of the epiphysial disc, very painful, and extremely sensitive to pressure. The child shrieks when it is moved. The disease occurs most often about the knees, and is usually multiple. It affects in civilization, artificially fed infants, especially those fed on sterilized foods. Other signs of scurvy, such as bleeding gums, are usually present. Scurvy recovers promptly on a proper diet, coupled with the administration of raw fruit juice.

The arthritides secondary to a diplostreptococcic infection in the tonsils and the deep urethra are almost always multiple, and react to a cleaning up of the original focus. Often they show an involvement of the synovial membrane out of all proportion to that of the bone, and may lack the slow and more or less steady progression of tuberculosis. The joints of the fingers are often attacked. Abscess formation is practically unknown in them, and they are rare in children. Still's disease is a disease of childhood but is multiarticular, and has an enlarged liver and spleen and characteristic blood changes.

Typhoid arthritis is almost always acute, and except in the spine, may be painless.

A differentiation of tuberculous arthritis from that of coccidioidal granuloma, is impossible without the detection of the causal organism; in tuberculosis, the tubercle bacillus, in coccidioidal granuloma, the *oïdium coccidioides*.

The differentiation from the second great type of arthritis is comparatively easy. The latter is essentially a disease of middle and late life. The joint creaks and grinds, and the obstruction to motion is mechanical; no

union is ever present between the articulating bones, except in the spine. Examination of the mouth will reveal root abscesses about the teeth. The positive diagnosis is made with the Röntgen rays. While the bone in both forms of arthritis shows rarefaction, the second type is peculiar in its ridges of bone at the lines of capsular attachment—the so-called lipping and spurring. In addition skiagrams of other joints usually show the disease in them, even when it is not manifest clinically. Of course, the guinea-pig test is negative.

Traumatic arthritis gives a definite history of a distinct trauma immediately precedent to the onset of the disease. An arthritis secondary to a capsular tear—a sprain—tends slowly to recovery, whereas a fracture is revealed by the Röntgen rays. A so-called traumatic arthritis, with a negative skiagram, which lasts for any length of time, especially one which slowly grows worse, is to be viewed with suspicion. Here again the guinea-pig will come to our aid.

New growths.—Benign neoplasms in the joints are very rare. It may almost be said that they do not occur.

Malignant growths.—Carcinomata are always secondary, and while observed occasionally in the spine, are rare in the joints of the extremities. They affect sometimes the marrow of one bone, but do not involve the joint, or the other bone of the articulation. A careful search will reveal the primary growth elsewhere.

Sarcoma of bone has no tendency to involve the joint. Sarcoma of the synovial membrane is a great rarity and can only be diagnosed by an examination of a piece of the tissue under the microscope.

The so-called giant cell sarcoma, or more properly benign giant cell growth, is fairly frequent in the ends of

the long bones of young adults, especially in the tibia. It is encapsulated, circumscribed, covered with a thin shell of bone, elastic, crackles like an egg shell, and shows no tendency to involve the joint. At operation it bleeds profusely, and is found to consist of the characteristic more or less crumbly granulation tissue, with currant jelly color, perhaps with streaks of yellow. A frozen section reveals the giant cells and the fibrous tissue, with evidences of old hemorrhages.

Finally a word of caution may be in order. Let it be emphasized again that a positive diagnosis cannot be made by a clinical examination. This statement may arouse contradiction, but it is based, not upon theory, but upon a mass of evidence. I have in my possession specimens of many joints operated on by some of the best men in the country.³¹ An examination of these specimens, and a study of the histories of the patients from whom they were taken, shows an astounding proportion of erroneous diagnoses. Many forms of treatment loudly heralded in the past are based upon the unsupported clinical opinion of their originators. If, on the other hand we admit our inability to arrive at a positive conclusion without the aid of the microscope, we are on the road to a correct handling of our cases.

In a general way little can be lost by waiting, before proceeding to radical treatment, especially if the joint be kept at rest with a splint, and it is far better to remove a pair of innocent tonsils from a patient with a tuberculous joint, or to give iodides and mercury for a few weeks, than it is to resect a joint that is the seat of an inflammation secondary to a mild diplostreptococcic infection in the tonsil, or to a syphilitic one in the neighboring bone marrow.

³¹ ELY, LEONARD W.: "Joint tuberculosis." William Wood and Co., 1911.

PROGNOSIS.—This is, generally speaking, good. A tuberculous joint, in itself, does not involve the patient's constitution, and is without danger unless in the neighborhood of vital structures, as for instance, the joints of the spine. Two things we fear: first, secondary infection, and, second, tuberculosis of other organs. Secondary infection immediately makes the prognosis grave, with its attendant general septic absorption, and its subsequent amyloid disease, and is to be avoided sedulously. The second danger is in the occurrence of tuberculosis of vital organs. The presence of a joint tuberculosis indicates a vulnerability to the tubercle bacillus and also the presence of a previous focus in the body. Statistics show that a large proportion of children with tuberculous joints never reach maturity, dying of the results of secondary infection or of tuberculosis of the lungs, meninges, etc.

The various joints carry different percentages of mortality, but modern methods bid fair to change the old statistics materially. Formerly spinal tuberculosis in the adult was practically invariably fatal. Now it is distinctly curable. Perhaps tuberculosis of the sacroiliac joint is the most dangerous of all, but this is fortunately rare.

TREATMENT.—As in other aspects of the disease, so in its treatment, the widest difference of opinion prevails. One must have some definite ideas in order to treat a case successfully, and, prefacing the subject with the admission that the theories and facts I am about to set forth, have not been generally accepted, I purpose laying down a very definite scheme of treatment. It is based not only upon clinical experience but also upon many years of laboratory work. We shall consider other forms of treatment afterward.

We have seen in our study of the pathology of joint tuberculosis, that the uncomplicated disease is strictly confined to the lymphoid marrow, and to the synovial membrane, and that it only attacks other tissues when a secondary infection is added. We have seen that tuberculosis of the joints is in itself a comparatively harmless disease, and only becomes very serious when a pus infection complicates it. We have seen further, not only in our study of the pathology, but also in that of the symptomatology, that all nature's efforts at cure seem to be expended in the direction of putting the joint at rest. These efforts are rarely completely successful, and nature is rarely able to effect a cure unaided, after the original marrow tuberculosis has spread far enough to involve the joint. Again, in the long run, viewed not only with reference to nature's local efforts at cure, but also with reference to her reaction to the tuberculous infection elsewhere in the body, it is evident that the final outcome depends upon the resistance of the patient.

Upon these observations we base our whole management of the case, and from them we deduce our three great rules of treatment, *viz.*:

1. Improve the patient's nutrition.
2. Avoid and prevent secondary infection.
3. Deprive the joint of function.

CONSTITUTIONAL TREATMENT.—Under this head would come the care of the patient's teeth and his digestion. He should have plenty of good, nutritious food, and the dairy from which he gets his milk should be free from suspicion of tuberculosis. Diseased tonsils should be removed. Occasionally tuberculous nodules will be found in them.

Adenoids also should be removed, on account of their influence upon the general health.

The patient should be in the open air as much as possible. If a child, he should stay out of school, at least for a while. Insanitary surroundings must not be tolerated. This applies especially to a habitation suspected of infection from other cases of tuberculosis.

As to climate, it is hard to speak positively. The disease has a wide distribution. A few years ago the seashore was supposed to be specific, now it is more or less displaced by the mountains. Rollier, of Leysin, Switzerland, has recently called attention again and forcibly to the healing powers of sunlight, and maintains that the sun's rays in Leysin possess peculiar therapeutic virtues.³² The patient's skin is exposed to the direct rays of the sun for as much of the twenty-four hours as possible. The exposure is to be begun gradually, the first day one arm for a half hour, the second day that arm for an hour and the other arm for a half hour, etc. This is continued until the patient lies on his frame, or goes about during the entire day, with nothing on except a hat and a breech clout.

While many observers have found that their patients do well when exposed to sunlight, no direct action of the sun's rays upon the tubercle bacilli in the bone marrow ever has been proved. Laboratory animals might easily decide the question. Until such proof is forthcoming we shall do well not to abandon local measures.

PREVENTION OF SECONDARY INFECTION.—All operations on tuberculous joints, even a simple aspiration, must be done with scrupulous asepsis. Every operation

³² ROLLIER, AUGUSTE: "Die Heliotherapie der Tuberkulose mit besonderer Berücksichtigung ihrer chirurgischen Formen." Berlin, J. Springer, 1913.

must have a definite purpose, and after it the wound must be closed. In no circumstances must a path be left open between the tuberculous focus and the outside air, for we know that infection almost inevitably will be established throughout its length. If we attempt to leave an exit for the tuberculous material, we shall really provide an entrance for pus germs. Tuberculous joints should not be scraped and packed. It is folly to insert a curette into them and blindly to scrape them out.

Cold abscesses, if deeply seated, small in size, not rapidly growing larger, nor causing severe pressure symptoms, may be let alone. Otherwise they should be aspirated under strict asepsis, repeatedly if necessary. Some surgeons simply apply pressure after the aspiration, others inject the abscess cavity with various substances, one of the most popular of which is iodoform, in mixtures or solution.

Calot's formula is olive oil 70 gr., ether 30 gr., creosote 5 gr., guaiacol 1 gr., and iodoform 10 gr. Two to ten grams of this mixture are injected into the abscess, after it has been evacuated. A 10 per cent. emulsion of iodoform, in glycerin or olive oil, may be tried.

In aspirating a cold abscess a very large needle or even a trocar is necessary. Otherwise the flocculi of broken down tissue will clog the needle and stop the flow. The aspiration is easier, if the skin and fascia are first slit with a scalpel.

Instead of aspirating the abscess, some surgeons open it freely, empty it, and immediately suture the wound carefully. The wisdom of wiping out the cavity is open to debate. To scrape its walls, and thereby to cause hemorrhage into it, is to invite trouble.

LOCAL TREATMENT

From our study of the anatomy, physiology and pathology of bones and joints we start our treatment with the following facts and theories:

1. Tuberculosis, when uncomplicated, is strictly a disease of the synovial membrane and of the lymphoid marrow. All the other tissues are immune, or practically so, to the direct infection, and suffer simply in the disturbance of their nutrition.

2. If these two tissues disappear, the disease dies out, or remains innocuous. It dies out because it has no pabulum, no culture material. It is starved out.

3. In spite of its subsidence, the tubercle bacilli may remain *in situ* indefinitely, ready at any time to start up the disease afresh, if conditions favorable to their growth are restored.

4. The presence of lymphoid marrow and synovial membrane in the region of the joint in the adult, is dependent upon function. If function be destroyed these two tissues disappear. In children lymphoid marrow exists in the shafts as well as in the bone ends, and its presence is not affected by function in the joints.

5. Nature's efforts at cure are all directed toward destroying the joint function, but unaided she rarely accomplishes this completely.

6. Operations on children's joints, by their interference with the centres of bone growth, cause marked crippling and shortening. The general opinion is that even when they are carefully done they are not so apt to be curative as operations on the joints of adults.

7. Conservative treatment in children is more likely to be followed by a state approaching cure than it is in adults. Some surgeons maintain that an actual cure with good

function can be attained in children by conservative treatment. Others, of which the author is one, consider that a cure sometimes can be attained thus, but rarely with any painless function. Conservative treatment in both children and adults, requires years of careful attention, and is almost impossible to carry out to the end in adults. Further, there is no absolute way of knowing when the joint is well.

8. No method of eradicating all the tuberculous tissue from about the joint has ever been devised, except amputation of the extremity well above the seat of the disease. Amputation, on the other hand, is hardly justifiable.

9. In spite of the impossibility of removing all the tuberculous tissue, we know that resections in the adult, when properly performed, are often followed by cure if they destroy joint function. On the other hand, no matter how much tuberculous tissue may be removed, if function remains the disease progresses.

10. The joints which are easiest to destroy by resection are those most easily cured by this means.

11. It is not absolute immobilization which always is responsible for the cure after a resection. When the head of the femur is removed, and the stump dislocates upward on the dorsum of the ilium, cure often results with a movable femur.

From these facts and theories we draw our great rule for local treatment—*Deprive the joint of function.*

This rule has two corollaries:

1. In children the treatment is always conservative. Conservative treatment should be followed in them until all hope of cure is gone, then amputation should be practised. No operation should be done which opens up the

diseased area. This rule does not apply to ankylosing operations on the spine, which are really done on bone at a distance from the tuberculous process.

2. In adults, the treatment is practically always radical, and its sole purpose is to destroy permanently all function in the joint, and thus to cause such a change in its structure as will render it an unsuitable habitation for the disease.

This second rule, it must be said, has not received by any means universal acceptance; in fact the great majority of surgeons carry out treatment on entirely different principles.

In operating on tuberculous joints the weight of opinion, numerically considered, favors the removal of as much tuberculous tissue as possible, and disregards completely the idea of destroying the joint. Some surgeons still open and pack tuberculous joints, but fortunately this practice gradually is losing favor. Some, on the theory of removing all the infected tissue, advocate amputation in disease of the joints of the extremities. While perhaps they may seem to follow the logical course, most authorities regard the treatment as more radical than necessary. In some quarters the practice of scraping out tuberculous joints still flourishes. It is mentioned here only to be condemned. The details of operative treatment will be taken up under the head of the various joints.

CONSERVATIVE TREATMENT

Under the head of conservative treatment, many expedients have been advocated, and some of these have survived. They are employed by many surgeons as adjuncts, and by some surgeons as their chief means of treatment. Among these may be mentioned passive hyperæmia,

injections of various sorts, internal medication, external applications, tuberculin, the Röntgen rays, etc.

The method of treatment which has obtained the widest recognition, and has established itself more or less generally throughout the profession, is that by rest, or in other words, by the deprivation of function. To this, in the case of the hip and knee, some surgeons add traction to draw the joint surfaces apart.

THE REST TREATMENT

This is carried out generally in three ways, *viz.*: By recumbency, by metal splints or "braces," and by plaster of Paris dressings, or "casts," as they are usually but erroneously called. The first is useful, especially in the spinal disease of children, when reinforced by a frame, and to meet special indications in the treatment of other joints in children and in adults, as will appear later.

For the manufacture of braces, a trained brace maker is desirable, but by no means essential, if the surgeon knows what he wishes to accomplish. It is far better to carry out the plan of a simple brace with the aid of the local blacksmith and harness maker, than it is to send a patient to a distant brace maker to be furnished with any sort of apparatus the latter may choose to supply. Again certain forms of braces are more or less standard, and for these the surgeon, with a little practice, can take his own measurements, and send them to a distant brace maker to be filled. In some cases a cast can be made of the limb, and this can be sent to the brace maker with directions.

In the past elaborate and complicated braces were the fashion, many of them designed to carry out some idea which their inventor considered important, but the modern trend is toward simplicity. Having mastered the funda-

mental principles of the treatment, a surgeon with a little mechanical bent is quite capable of carrying it through.

In the first place, in the treatment of tuberculous joints, apparatus should not be employed to correct deformity. The deformity is corrected first and then the apparatus is applied, or, especially in the case of the hip joint, the apparatus is applied, and then the patient is put to bed and traction is made on the apparatus in the line of deformity until the deformity is reduced. With plaster of Paris, the deformity is partly reduced before the plaster is put on, and the correction is continued with each succeeding application of plaster.

With either a brace or a plaster dressing, the deformity may be corrected under ether before treatment is begun. This is the quickest and in some respects the most satisfactory method, but it requires great care, on account of the danger of stirring up the disease by the manipulation. Even then the method is not free from danger, and some surgeons refuse to employ it on that account.

Nothing is to be expected from treatment by braces or by plaster in a few weeks, or months. It must be maintained continuously for a minimum of one year, and often for many years. If a brace gets out of order, it must be immediately repaired, and this is one of the disadvantages of the brace treatment as compared with that by plaster of Paris. Another disadvantage lies in the ability of the patient to loosen his apparatus. Children will always do this if they can. A third disadvantage, and perhaps the greatest, unless the surgeon has his own instrument shop, is that in a treatment which depends for its success upon attention to details, the carrying out of the details is in a

large measure not in the surgeon's hands but in those of a mechanic.

The advantages of braces over plaster are cleanliness and the ready accessibility of the joint.

Plaster of Paris, or one of its substitutes, forms a satisfactory means of immobilization, if care be used and if skill be cultivated in its application. All bony prominences under the dressing must be well padded; muscle bellies need no padding. Sheet wadding, the so-called interlining, makes a good padding, as does piano felting, or silence cloth. Saddler's felt may be employed, but is rather coarse and heavy. A muslin or gauze bandage covers the limb, or the limb and part of the trunk, snugly and smoothly but not too tightly. The plaster bandages are wrung out of warm water, and are evenly applied until the dressing is strong enough to withstand the strain to which it will be exposed, but not heavy enough to be cumbersome. It may be reinforced by extra turns in the region of the joints, where the strain comes, or metal or thin wooden splints may be incorporated in it. A short plaster dressing, reaching a few inches above and below the affected joint, is of little use. It should be long enough practically to immobilize the joint, and this means, for instance, in a joint like the knee, that it should extend from the perineum to the malleoli, in the ankle, from the toes to the proximal end of the leg, etc.

If one does a great deal of plaster work one will find the so-called stockinette a good material to have on hand in various sizes. It is drawn on the part to be bandaged, next to the skin. It not only makes the inner surface of the dressing smooth, but, if it be cut very long, it can be

turned down over the outside of the plaster, and will serve to protect the dressing and to keep it clean. The "scratch" bandage is a muslin or linen bandage which is slipped under the stockinette. It is drawn up and down every day, and keeps the skin in good condition, especially if dusting powder be liberally used.

One of the disadvantages of plaster is the fact that it may cause pressure sores. Pressure sores are almost always the result of improper padding. They make their presence known almost immediately by a foul odor, once smelled never forgotten, and necessitate the immediate removal of the dressing.

Good plaster bandages can be bought in the shops, but the best are homemade. The knack is easily cultivated. Crinoline of a good brand, containing starch but no glue, is torn into strips about five yards long. Four and six inches are good standard widths. Some surgeons prefer unstarched crinoline, some employ gauze. One rubs by hand the finest quality of dental plaster into the meshes of a strip of crinoline, rolling the bandage loosely meanwhile, and using just as much plaster as the meshes of the crinoline will take up, and no more. If each bandage is wrapped in paper, and all are kept in a tight tin can, they will keep for a long time without deteriorating.

To apply a plaster dressing one stands a bandage on end in a pail about two thirds full of warm water, and leaves it there until the bubbles cease to rise. The next bandage is then put into the pail, and the first one is removed, wrung out lightly, and applied quickly, smoothly and fairly snugly. Reverses are not used. The second one is ready when the first one is on. The third one is put

in the water before the second is taken out. Otherwise wet hands scatter drops of water into the pail of plaster bandages. If the bandages have been loosely rolled, and if a hole have been left down through the centre of each, the water permeates the whole quickly, and no delay occurs.

The dressing extends, as it is put on, well above and below the distance intended for the finished dressing, and the superfluous portions are cut away afterward with a sharp knife. A good knife for this purpose is an ordinary pruning knife whose point has been ground away, though a jackknife or a potato knife will serve well. A rough edge, a wire edge, such as can be put on with a file or a coarse stone, is better than a smooth, fine edge. The dressing is best trimmed when it is finished. The trimming is accomplished by a combination of tear by a steady pull with one hand, and a cut by a stroke of the knife in the other.

If the proper materials have been employed, no salt or any other substance is necessary or advisable in the water to hasten the setting of the plaster. The dressing should be set enough to hold well by the time it is finished. If it is not, then something is the matter with the materials out of which it is made. Complete setting usually takes about 48 hours. Until the end of that time, the dressing should be exposed to the air, and should not be subjected to undue pressure or strain.

A good plaster dressing, if not exposed to blows, and if kept clean and dry, will last almost indefinitely. The life of dressings may be said to be proportional to their cost. I have seen them firm and beautiful after a year—in pay patients. They are often filthy and soft after a week or two, when they have been provided gratis.

JOINT TUBERCULOSIS IN GENERAL

OTHER NON-OPERATIVE MEASURES

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PASSIVE HYPERÆMIA³³

This treatment, while by no means new, was revived and recommended for joint tuberculosis, by Bier of Germany, and often it is called the Bier treatment on that account. It consists in producing a passive congestion in a limb, usually by means of an Esmarch bandage. The bandage is applied well proximal to the affected joint, just tightly enough to make the extremity distal to it dusky red and warm, not blue and cold, and is left on at first about fifteen minutes daily. The time is gradually extended until the bandage remains on two or three hours. In cases with abscess special cupping glasses have been devised to treat the abscess by suction. Some surgeons have had good results with this treatment, in the hands of others it has proved a disappointment.

TREATMENT BY INJECTIONS³⁴

The first recommendation of this treatment I have been able to find is in an article by von Mikulicz in 1881.³⁵ Von Mikulicz employed iodoform, and this substance has since been employed more widely than any other. Many others have also been tried, and some have been loudly praised. Among the number may be mentioned bone charcoal, iodine, phenol, arsenious acid and corrosive sublimate,

³³ BIER, A.: "Ueber ein neues Verfahren der conservativen Behandlung von Gelenktuberkulose." *Ver. d. deut. Gesell. f. Chir.*, 1892, i, 91.

BIER, A.: "Hyperæmie als Heilmittel." Leipzig, F. C. W. Vogel, 1907.

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³⁴ ELY, LEONARD W.: "The injection treatment of tuberculous joints." *J. Am. Med. Ass.*, 1913, lxi, 1453.

³⁵ VON MIKULICZ: "Ueber das Iodoform als Verbandmittel bei Knochen- und Gelenktuberculose." *Berliner klin. Woch.*, 1881, xviii, 230.

acidulated solution of calcium sulphate, zinc chloride, balsam of Peru, naphthol camphor, and formaldehyde solution. Some of these substances are by nature germicidal, but others are not. The surgeon who would try the treatment of tuberculous joints by injection should assure himself that the substance to be injected is at least sterile. Iodoform may be used in a 1-10 solution in ether, or as an emulsion.

BECK'S PASTE.³⁶—This paste, consisting of bismuth subnitrate 1 part and vaseline 2 parts, sometimes with white wax added, has had quite a vogue, especially in cases with infected sinuses. The treatment has caused several deaths from bismuth poisoning, and should be employed with great circumspection. Beck emphasizes the danger of using it in the presence of a sequestrum. In the effort to gain the mechanical effect of the paste without the dangers of the bismuth ingredient other surgeons have recommended substitutes. While it is doubtful that any of these pastes have a specific effect upon the tubercle bacilli, it is possible that they act mechanically to check the secondary infection, and so convert the disease into a simple, uncomplicated joint tuberculosis.

TUBERCULIN TREATMENT

This treatment was quite popular a few years ago, and is still advocated by some surgeons, especially when combined with other measures, such as rest.

THE RÖNTGEN RAYS

Theoretically this should be an almost ideal form of treatment, on account of the action of the rays upon lymphoid tissue. Practically the results of treatment have not

³⁶ BECK, EMIL G.: "Bismuth paste in chronic suppurations." St. Louis. C. V. Mosby. Co., 1915.

justified the expectations that were entertained of it. Perhaps increasing knowledge of this therapeutic agent may teach a way to employ it more successfully.

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SECTION V.

TUBERCULOSIS OF SPECIAL JOINTS

CHAPTER I.

TUBERCULOSIS OF THE SPINE, POTT'S DISEASE

THE disease affects primarily, principally, and almost invariably, the marrow of the vertebral bodies. The pedicles, laminae and lateral masses, composed as they are of dense bone, escape. The lateral articulations seldom if ever are involved. The starting point seems usually to be near the anterior portion of the body. The disease may be confined to one vertebra, but usually attacks several, spreading from one to the other beneath the anterior ligament. The intervertebral disc probably escapes until the bone in its immediate vicinity is damaged. The whole body may be attacked, or its anterior portion exclusively. Sometimes the process involves the anterior part of several vertebrae, leaving the rest unaffected.

As the marrow of the bodies becomes more and more involved, the bone is killed, the body softens, and then collapses slowly, at first in its anterior portion, thus taking on the shape of a wedge with the point forward. Later the entire body may be destroyed. As the body collapses, the superimposed weight is transferred to the lateral masses, and in extreme cases perhaps even to the spinous processes and laminae. As this takes place, the spine above the lesion slowly falls forward, causing a protrusion backward of the spinous processes at the site of the disease—the well-known kyphosis, buckle or hump. At first, this kyphosis is almost invariably angular, but in the later stages of the disease it tends to become rounded.

The vertebrae, becoming involved one after another, soften and collapse, the weight of the trunk above bending

the spine more and more forward to fill in the space left by their destruction. This causes a steady growth in the angular kyphosis, accelerated in the thoracic region by the

normal kyphosis there, but retarded in the lumbar region by the normal lumbar lordosis. Spontaneous cure may be effected at length by an ankylosis of the spinous processes after an enormous amount of deformity has taken place, but a complete fusion is probably very rare. Occasionally at operation on old cases, however, evidences of bony union here and there are unearthed.

As the bodies collapse, the necrotic material is squeezed out anteriorly, and collects under the anterior ligament as a fusiform mass visible in the Röntgen plate. It may remain there indefinitely as a cold abscess, or at any time it may rupture the anterior ligament, and start on its journey to the surface, its course determined by gravity and the direction of least resistance. In high cervical disease it appears in the back of the throat as a post-pharyngeal abscess; in disease lower down, as a fluctuating

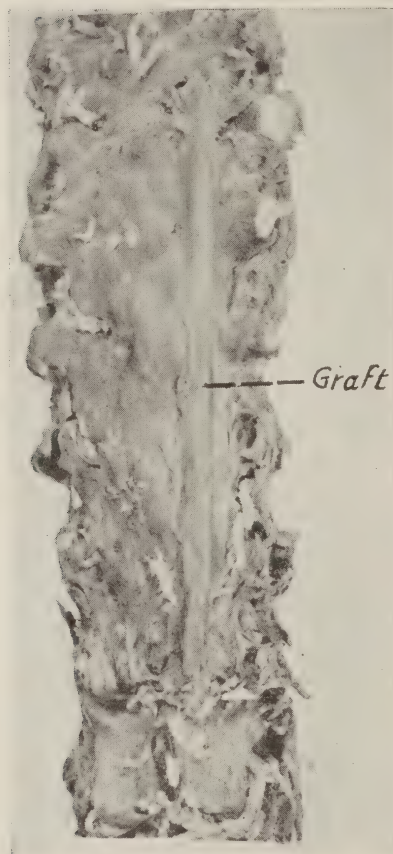


FIG. 73.—Posterior aspect of tuberculous spine of child, upon which a Hibbs operation was done. The symptoms persisted, and at a later operation a bone graft was laid down on the laminae. The patient died three months later of tuberculous meningitis.

swelling at the side of the neck. In thoracic disease the abscess lies at first in the posterior mediastinum, then,

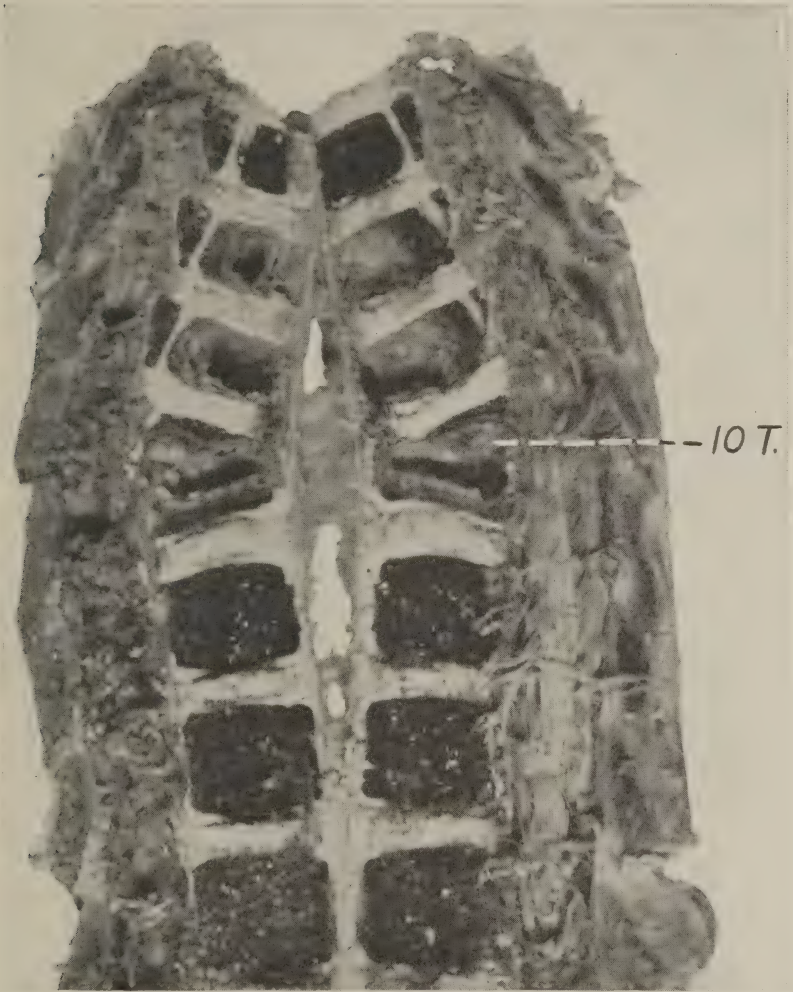


FIG. 74.—Spine shown in preceding figure, laid open from behind. Note the wedge shape of the vertebra first involved. Note also that although the two vertebrae above are extensively involved, they have been kept from collapsing by the bony bridge in the laminae

making its way behind the diaphragm, it gains the sheath of the psoas muscle, and, following this downward under

Poupart's ligament, comes through the saphenous opening, and appears finally as a fluctuating swelling on the antero-medial aspect of the thigh. In rare instances a cold abscess makes its way through Petit's triangle, and appears in the back.

The tuberculous material is squeezed out not only

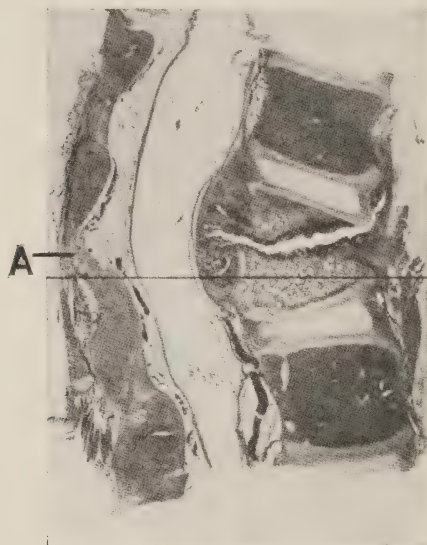


FIG. 75.—Tuberculosis of the spine. Photograph of the stained slide, sagittal section. The child died five months after a Hibbs operation, from an intercurrent disease. Bony union of spinous processes at A. Note wedge shape of affected vertebra, with apex forward, and the indication of the beginning of a cold abscess anteriorly. When one looks at the mass of tuberculous granulations on the posterior surface of the body of the vertebra, pushing out into the spinal canal, one can readily understand the pathogenesis of Pott's paraplegia. The marked calcification in the diseased vertebra shows that healing was in progress.

anteriorly, but also posteriorly, where it accumulates between the vertebral body and the dura. If it attain proportions great enough, it exerts pressure upon the anterior columns of the cord, and causes paralysis in the parts below the lesion, the so-called Pott's paraplegia. Even in

cases with marked deformity it is doubtful if this paralysis ever is caused by bony pressure. It is most frequent in cervical disease. When the cold abscess breaks through the spinal membrane it causes a tuberculous meningitis.¹

In spite of extensive destruction of bone, the spinal foramina are not compromised in their lumen. Pressure upon the spinal nerves as they pass through them probably never occurs.

In high cervical disease dislocation may take place, with instant death from damage to the phrenic nerve. In thoracic and in lumbar disease with much deformity, the viscera are distorted, displaced, and often compromised in their function. Whether from this cause or not, there is a peculiar facies of old hunchbacks that can be recognized at a glance. The neck is short, the shoulders are broad, the arms appear abnormally long, and the trunk is short. This last factor accentuates the diminutive size of these hunchbacks when they sit down.

SYMPTOMATOLOGY

Pain is usually the most pronounced symptom. It varies in intensity from a slight feeling of discomfort or stiffness to great severity. It may be more or less constant, or it may intermit. Frequently it comes in paroxysms, especially in a child. Evidently as the result of an unguarded movement the child suddenly doubles up, screaming with pain. In the early stages of the disease the pain is more in the nature of a feeling of stiffness, noticed for a while in the morning after rising, and wearing away with exercise. This also occurs especially in the child, and is temporary. The pain of a tuberculous joint is almost always aggravated by motion.

¹ ZIEGLER, ERNST: "Lehrbuch der speziellen pathologischen Anatomie." 11th. Auflage. Jena. Verlage von Gustav Fischer, 1906, Band ii, Seite 371.

The pain may be located in the back, at or near the lesion, or at a distance from it. At some period of the disease pain in the back will be experienced, but it may be insignificant in comparison with that felt out along the course of the spinal nerves—the so-called referred pain of Pott's disease. This pain varies of course according to the location of the disease. In the cervical spine it may be felt in the neck, or in the arms as a brachial neuralgia, a "neuritis." In the thoracic spine it radiates around the trunk as an intercostal neuralgia, or "muscular rheumatism." In disease of this region the patient often exhibits a peculiar grunting respiration, or a futile spasmodic cough.

In low thoracic or in lumbar disease a typical "girdle pain" may be present. In lumbar involvement the patient may complain of pain along the anterior crural or the sciatic nerve. In adults a persistent sciatica, often double, may for months be the only sign of trouble. In women the pain in the back and in the abdomen may be mistaken for that of a uterine displacement or perineal laceration, and, in either sex, for that of an appendicitis.

These so-called referred pains are usually ascribed to irritation of the spinal nerves as they pass through the spinal foramina, but we have seen that this cannot be so, for the foramina remain patent and unchanged. The pain is a true referred pain, similar to that occasionally experienced in the foot after it has been amputated, or in the knee with hip joint disease. It does not differ essentially from the referred pains of tabes. When a painful stimulus comes in over a nerve from an inflamed spinal joint, it is referred to the paths over which painful stimuli usually come in at that level.

The muscular spasm which is so characteristic of the disease holds the joints of the spine more or less at rest.

When it relaxes in sleep, the joint moves, and the child wakes with a cry—the so-called night cry, heard more often with Pott's disease than with disease of the other joints. When the mother reaches the child's bed she often finds it asleep.

The muscular spasm is responsible also not only for the feeling of stiffness of which the patient complains, but also for the very evident appearance of stiffness which he presents, and for the limitation of motion of a greater or smaller segment of the spine. This is nature's involuntary protective mechanism, and it is soon reinforced by the voluntary efforts of the patient. He holds himself stiffly, and avoids jars. Often he adopts an attitude almost characteristic of disease of a certain part of the spine. Thus in high cervical disease, the head may be acutely flexed, bringing the chin down on the sternum. When the disease is somewhat lower the head may be thrown forward or back, or it may be tilted to the side in one of a number of different attitudes. In thoracic disease the shoulders may have an exaggerated appearance of squareness. In lumbar disease the trunk is held in superextension, and the abdomen is protruded. Both in thoracic and in lumbar disease the patient, especially if he be a child, will often support himself with his hands while sitting.

It is seen therefore that there is nothing characteristic of the attitude of a tuberculous spine, but that it varies with the region involved, and may be described simply as a departure from the normal.

Muscular spasm causes also limitation of motion, usually more marked in flexion than in extension. Muscular spasm and limitation of motion really go hand in hand. Each may be said to cause the other. The limitation is not general, but affects that portion of the back where the

disease is located. It is very rarely absent, but is somewhat harder to detect in the thoracic than in the cervical or lumbar spine. To elicit it, one directs the patient to put the various regions of the spine through their normal range of motion, and notes any restriction.

In the examination of the cervical spine one should bear in mind that nodding of the head takes place at the occipito-atlantoid articulation, rotation at the atlanto-axoid, and flexion, extension and lateral motion in the other cervical articulations. In disease of the lumbar spine, when the patient is directed to pick up an object from the floor, he will not bend forward naturally, with a flexed spine, but, with his trunk held rigidly upright, he will flex his hips and his knees, and pick the object up at his side. Each region has its peculiar stiffness, but wherever the lesion may be, the muscular spasm is responsible for a general appearance of stiffness and awkwardness, often to be easily noticed as the patient moves about. Cases of Pott's disease without limitation of motion are very rare, but they do occur.

The characteristic deformity of Pott's disease is a kyphosis, a curve or angulation with posterior convexity. It is wont to appear earlier, and to be more noticeable in the thoracic region, where the spine has a normal backward convexity, than in the lumbar, where the convexity is forward. It usually appears earlier in children than in adults. In the lumbar spine of the adult it may be absent indefinitely. At first it is distinctly angulated—a knuckle—but later, with the involvement of several vertebræ, it becomes rounded off. On the other hand, in adults, the kyphosis may be rounded from the start, and in the thoracic region may constitute simply an exaggeration of the normal thoracic curve. In early lumbar disease the kyphosis may be

represented by a flattening, which replaces the normal lumbar lordosis.

A lateral curvature may be added to the kyphosis, or, occasionally, and less often in children than in adults, it may be the first deformity to appear. It is said to be caused by the greater involvement of one side of the column, or to disease in a lateral articulation.

Above and below the knuckle in early cases, the spine is usually flattened. Generally speaking, a change in the curve in any region of the spine must be compensated by change in contour of other regions, and in old case of Pott's disease with marked kyphosis, lordosis, equal or nearly equal will be present elsewhere, accentuating the deformity. This compensatory lordosis helps to differentiate spinal tuberculosis from other forms of spinal arthritis. In these it is usually not present.

Cold abscess formation is very frequent in spinal tuberculosis, but on account of its distance from the surface,

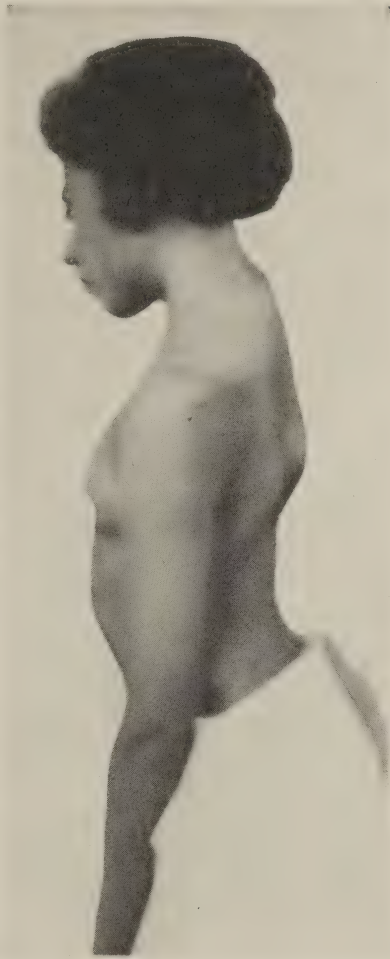


FIG. 76.—Tuberculosis of the cervico-thoracic spine, treated intermittently with jackets for nine years.

the abscess, if of small size often escapes recognition. It need cause no symptoms whatever. If it increase in size, it

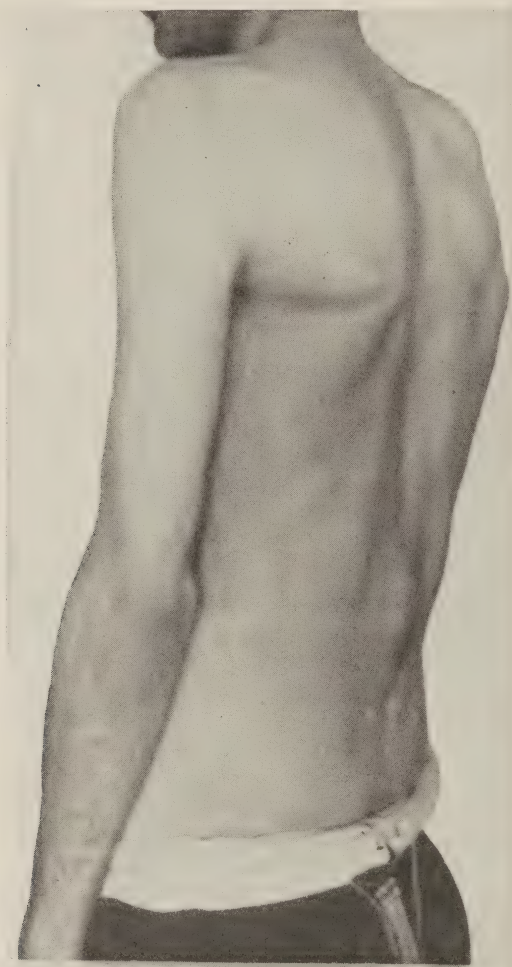


FIG. 77.—Tuberculosis of the lumbar spine. Albee operation. The patient was unruly after the operation, and the disease was not arrested.

may cause pressure symptoms upon contiguous structures. An abscess in the cervical region may give rise to dysphagia, or to dyspnœa. When it comes from the upper part of the cervical spine, it is recognized as a fluctuating swelling in the posterior pharyngeal wall. Lower down it may appear as a fluctuating swelling in the side of the neck.

A cold abscess in the thoracic region is hard to detect clinically, but in the lumbar region it may be palpated as a deeply seated, tense, obscurely

fluctuating mass in the flank, lying on or in the sheath of the psoas muscle. The irritation of its presence causes

a spasm and a contraction of the psoas-iliacus muscle, recognized clinically by the fixed flexion deformity of the hip. If the patient continues to be about on his feet, the abscess follows along down the sheath of the muscle, becoming more and more superficial, passes under Poupart's ligament, and usually points on the antero-medial aspect of the thigh in the region of the saphenous opening. In exceptional cases it may pass through Petit's triangle, and point in the back, or through the sciatic foramen, and appear in the gluteal region.

When these cold abscesses become infected secondarily by pus germs, constitutional symptoms of septic absorption supervene, and a condition of chronic suppuration results, if the patient survive the acute stages, which may run on indefinitely. We can picture the state of affairs, in such a case, and cannot expect healing. At the bottom of a long, narrow and tortuous sinus, is a secondarily infected tuberculous bone

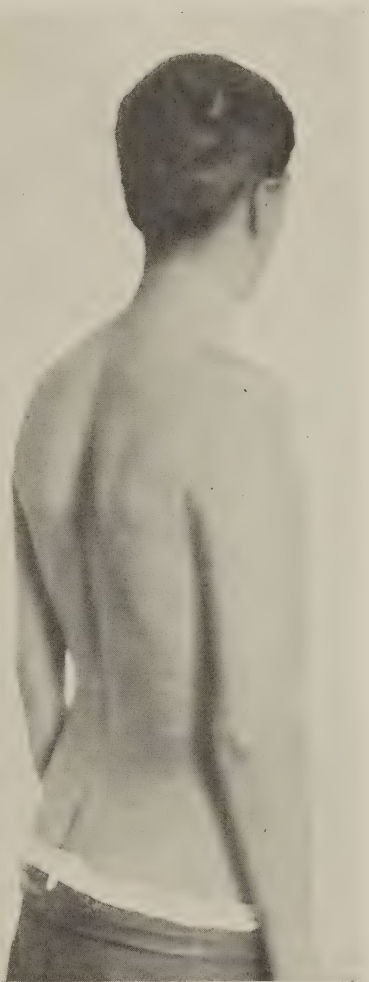


FIG. 78.—Tuberculosis of the second lumbar vertebra. Guinea pig test with contents of psoas abscess was positive. Hibb's operation in September, 1920; photograph taken nineteen months later. Note absence of deformity.

cavity, perhaps containing sequestra. Healing in such circumstances is highly improbable, unless we can obliterate the cavity, and remove the sequestra.

Pott's paraplegia is a compression myelitis caused by pressure on the anterior columns of the cord by the tuberculous inflammatory products beneath the posterior common ligament, seldom if ever by bony pressure. It is most often seen in disease of the cervical or of the upper thoracic spine. It is a spastic paralysis, affecting usually the lower extremities. It begins as a slight unsteadiness in walking, and sometimes progresses to complete motor paralysis. The knee jerks are increased, unless their centre in the cord be affected, when they may be absent. Ankle clonus is usually present. In severe cases the sphincters of the bladder and of the rectum may be involved, with resulting incontinence of urine and of fæces. Motion is affected much more than sensation, but anæsthesias or paræsthesias may be present.

In mild cases, properly treated, the paralysis may clear up in a short time, but usually it lasts for months. Occasionally it may be permanent. Sometimes a spastic paraplegia is the first thing to draw attention to the presence of a tuberculous lesion of the spine of an adult, and on the other hand many cases of Pott's paraplegia have been mistaken for essential cord lesions, with no thought for the bone lesion underlying them.²

DIAGNOSIS

This is made tentatively, in early cases, on the symptoms and physical signs, and confirmed by the Röntgen picture. The patient complains of pain and stiffness, usually of gradual onset and of some duration. In a child

² PAINTER, CHARLES F., AND MOORE, GEORGE C.: "Pott's paraplegia." *Am. J. Orth. Surg.*, 1910-11, viii. 306.

the trouble may have come on after an attack of measles, whooping cough, or scarlet fever. Examination shows a peculiar attitude, with a change in contour of the spine, usually in the shape of an angular kyphosis. Muscular spasm and limitation can be demonstrated by attempting to put the spine through its normal range of motion. Often a deep abscess can be palpated. The knee jerks may be changed. A positive test with old tuberculin may be of assistance.

The X-rays show a rarefaction of the vertebral body, usually with some compression, and a diminution of the space between the bodies.

DIFFERENTIAL DIAGNOSIS

From coccidioidal granuloma, the differentiation is impossible without the examination of some of the diseased tissue. The other members of the first great type which are most important to differentiate, are syphilitic arthritis, typhoid arthritis, and the arthritis caused probably by diplostreptococcic infection, supposed to be due to chronic infection in the tonsil or in the deep urethra.

Syphilitic arthritis in the spine is rather rare, doubly so in children. As a rule, a history of syphilis can be elicited, with a positive Wassermann test, and other signs of syphilis can be found. Muscular spasm, and limitation of motion are not so marked, the deformity is usually less, and the distinct kyphosis, the evidence of a crushed vertebra, is lacking. The wedge shaped crushing therefore is lacking in the X-ray plate also. Antisyphilitic treatment causes an improvement of the disease.

Typhoid spine is much more acute in its onset than tuberculosis, and occurs in the late stages of typhoid fever or during convalescence. The pain, stiffness and muscular

spasm are out of all proportion to the bony damage as revealed by deformity or the X-ray. A kyphosis is extremely rare in typhoid spine, and the Röntgen rays show little if any rarefaction. A bony ankylosis of two vertebræ makes one suspect typhoid spine.

In the chronic spinal arthritis caused by an infection in the tonsil or in the deep urethra, the involvement is not circumscribed as in tuberculosis, but more or less general. Often the entire spine, with the exception of the two uppermost joints, is affected. For some reason these usually escape. The affection seems to be largely in the soft parts of the joints; the stiffness and pain are wont to be out of all proportion to the damage in the bone. The X-rays do not show the extensive local destruction of bone, with the compression and coalescence of the vertebral bodies. Cold abscess formation does not occur in this form of arthritis, and a local kyphosis does not form.

In the second great type of spinal arthritis the stiffness and limitation may be more or less localized, and a slight angular kyphosis might possibly give rise to a suspicion of tuberculosis. Usually however there is no abnormal kyphosis, and the disease is more or less general in the spine, even though it may be more pronounced in a certain segment. The second type of chronic arthritis occurs usually in middle aged or elderly persons. The two diseases can be differentiated absolutely by the Röntgen rays. Spurring and lipping are present in the second type of chronic arthritis and are absent in tuberculosis.

Spinal fractures cause an angular deformity similar to that of Pott's disease. The diagnosis is to be made upon the distinct history of trauma followed immediately by the deformity and by other symptoms, especially by

symptoms of cord pressure such as incontinence of fæces and urine. Before the injury there must have been no symptoms referable to the spine. The Röntgen rays show crushing and distortion, often a broadening out of the body, rather than a rarefaction.

In *torticollis* such motions as put the muscles involved, usually the sterno-mastoid and trapezius upon the stretch, will be painful and limited, and the deformity will be typical of their contraction, whereas in tuberculosis of the cervical spine the muscular spasm and limitation of motion will be more general. Torticollis following trauma, is sometimes caused by a fracture of one of the cervical vertebrae, and can be detected by the Röntgen rays.

Rotary-lateral curvature is accompanied by little pain, if any. Severe cases may cause an ache or a tired feeling in the back. Lateral curvature is a postural deformity, not a disease; hence subjective symptoms will be slight or absent. The curve is not only lateral, but it has a twist also which throws the ribs into prominence on one side of the back (best seen when the patient leans forward) and makes one shoulder high or one hip prominent. The curve is often S-shaped. No knuckle or kyphosis is ever present on the spine, nor can muscular spasm be detected. The apex of the deformity is always on the ribs—also best seen when the patient bends forward.

Rickets.—In a young child with severe rickets, the continuous sitting posture necessitated by his weakness causes a long posterior curve, often with more or less angularity in the thoracic region. This curve may be somewhat fixed, but as a rule, if the child be laid prone, his hips be raised from the table, and the kyphosis be pressed in with the palm of the hand, the curve can be obliterated, or almost so. In addition, the child will present other

evidences of rickets. For some unknown reason, the combination of rickets and joint tuberculosis in the same patient, is extremely rare, if it ever occurs.

Sarcoma is more rapid in its course than tuberculosis, and causes a more diffuse, rounded, irregular deformity, not following the spinous processes, and sometimes sensitive to pressure. The Röntgen rays may show extensive destruction of bone, possibly combined with the formation of new bone. Sarcoma is very rare.

Carcinoma occurs as a metastasis of a growth elsewhere, and is accompanied by agonizing pain not relieved by rest. The Röntgen rays show an extensive destruction of the vertebral bodies but without the coalescence and wedge shape of tuberculosis. Careful search reveals the primary growth.

Hip disease is sometimes confused with the psoas contraction of Pott's disease. In the former all motions of the joint are limited, whereas in psoas contraction, if the psoas-iliacus be relaxed by further flexion of the thigh, the motions of the hip are free.

Persistent "*lumbago*" and "*sciatica*" are to be viewed with suspicion until the absence of a bony lesion has been demonstrated.

PROGNOSIS

Until recently, the prognosis in tuberculosis of the spine was bad, but modern methods of treatment have changed the outlook. Prognosis in simple Pott's disease now is good. Abscess formation makes the prognosis unfavorable. Disease of the cervical region, on account of the proximity of vital structures has an added danger. Under operative treatment Pott's disease can be brought to a standstill within six months. Without operation three

years may be said to be the minimum duration of treatment. Bony deformity once present, is permanent. Perhaps by strict attention to details and by vigorous measures, the hump may sometimes be made a trifle smaller. Under modern methods of treatment, the enormous humps, so frequently seen in former days, are growing rarer.

TREATMENT

The treatment of tuberculosis of the spine consists in immobilization, and this immobilization may be made complete, by operation, or relative, by apparatus. Realizing the importance of rest in the treatment of this disease many surgeons had attempted to devise an operation which would completely ankylose the spine at the seat of the disease. Hibbs^{3,4,5} was the first to solve the problem, getting his idea from the occasional cure wrought by nature through the fusion of the spinous processes. Albee followed Hibbs shortly.^{6,7,8} Various modifications have been made of these two operations, but they remain standard. Enough time has not elapsed for a final opinion as to their merits. Some surgeons refuse to employ them, and cling to the treatment by apparatus. Others, of whom the author is one, consider that they have practically displaced the old treatment, and employ them on every possible occasion. Others pursue

³ HIBBS, R. A.: "An operation for progressive spinal deformities." *N. Y. Med. J.*, 1911, xciii, 1013.

⁴ HIBBS, R. A.: "Operation for Pott's disease of the spine." *J. A. Med. Ass.*, 1912, lix, 433.

⁵ HIBBS, R. A.: "Treatment of vertebral tuberculosis by fusion operation." *J. A. M. A.*, 1918, lxxi, 1372.

⁶ ALBEE, F. H.: "Bone transplantation etc., in Pott's disease." *N. Y. Med. Jour.*, 1912, xcv, 469.

⁷ ALBEE, F. H.: "A study of 539 cases of Pott's disease, etc." *Am. J. Orth. Surg.*, 1916, xiv, 134.

⁸ ALBEE, F. H.: "Transplantation of a portion of the tibia into the spine for Pott's disease." *Journ. of the A. Med. Ass.*, 1911, lvii, 885.

a middle course. Each operation has its merits. The Albee is much the easier to perform, but the Hibbs seems much the more reliable and secure. Both are serious

operations, and should never be performed without the assistance of a skilled anæsthetist.⁹

THE HIBB'S OPERATION.—The patient lies prone, with pillows so disposed under his chest and shoulders, that his breathing, when deeply anæsthetized, will be unrestricted. The operator makes his incision in the mid-line through the skin and fascia, of such a length as will enable him to work upon the spinous processes of the diseased vertebræ and upon those of one or two vertebræ above and below them. Previ-

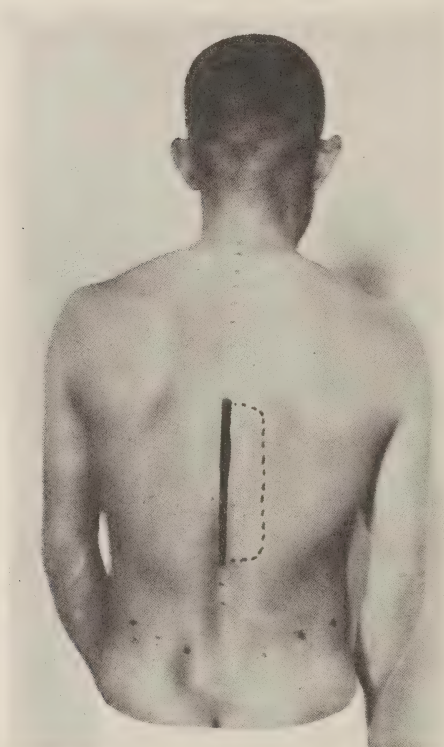


FIG. 79.—Incisions for the Hibbs and Albee operations. The solid line is the Hibbs incision, the dotted line the Albee.

ous to the operation, of course, the exact extent of the disease has been determined by the Röntgen rays. The skin is then shut off with towels. Hibbs towel clamps make this easy and rapid. From now on three rules will be found helpful:

⁹ ELY, LEONARD W.: "Ankylosing operations on the spine, etc." *Jour. A. M. A.*, 1917, lxxviii, 183.

1. Take plenty of time. Do not try to hurry, but do the dissection carefully.

2. In the first part of the work keep exactly in the mid-line, and so avoid hemorrhage. If this and the next rule be observed, hemostasis will not be necessary.

3. In all skeletonizing, keep close to the bone. The instant one gets away from it, one cuts blood vessels which must be tied.

The next step is to incise the periosteum over each spinous process, and to push it back for a short distance. Small pieces of gauze are then tucked tightly under the periosteum, checking bleeding and pushing the periosteum back still farther. The tips of all the spinous processes to be operated upon are to be laid bare before proceeding to the next step.

Insert a small scalpel close to the last spinous process, and divide the interspinous ligament between it and the spinous process next higher up, exactly in the mid-line. Divide in the same manner the interspinous ligament of all the spines to be operated upon, and dissect the periosteum from one side of the spinous processes, and from all the laminae on one side, packing the wound behind you with larger pieces of gauze as you go. The assistant in sponging aids the dissection by pushing the periosteum off the bone as he sponges. The dissection is carried laterally as far as the lateral articulations, whose location and plane have been ascertained on the skeleton if necessary, before the operation was started. Divide the ligament of each lateral articulation, and scrape it out thoroughly with a small curette. Repeat these procedures upon the other side. Be sure to remove every bit of periosteum from the spines and the laminae, and bare the upper and lower borders of the laminae of every vestige of the ligamenta

subflava, working a small curette even somewhat around on their anterior aspect. All this takes a great deal of

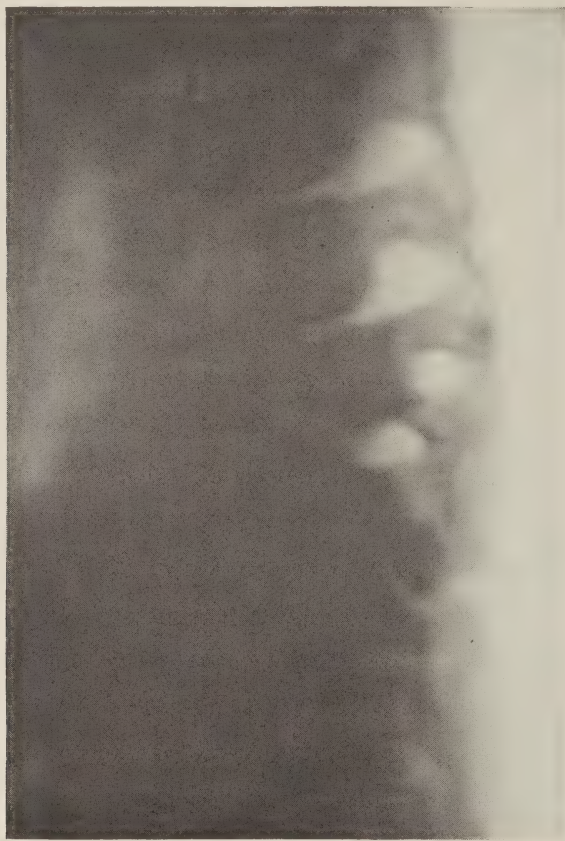


FIG. 80.—Tuberculosis of the spine. Crushing together of the bodies of the 3rd and 4th lumbar. Note their wedge shape. The disease in the stage of healing. A Hibbs operation was performed eighteen months previously. Union was not obtained between the 4th and 5th lumbar vertebræ, and a second Hibbs operation was done nine months later. The child now is apparently well. Note the bony bridge in the laminæ, and the fairly sharp outline of the two affected vertebræ.

time and some anatomical knowledge, and must be done deliberately. The rest is comparatively simple.

With a small Hibbs gouge or chisel inserted at the base of the spinous process, chisel a very thin wedge or sliver

of bone from the lower border of each lamina with its base lateral, leaving the base of the wedge intact, and, still attached at its base, turn this wedge or sliver down across the interlaminar space upon the next lamina below it. Hibbs now is varying this by chiseling a flake from the posterior surface of the lamina, and turning it upward on the lamina above.

With a pair of Hibbs bone forceps partially divide each spine at its base, leaving only its lower or distal portion intact, and break each spine down, so that its tip rests upon the raw base of the one below it.

With kangaroo tendon or ten day catgut, suture the periosteum and deep fascia in the form of a tube. This is best done in stages as the spines are fractured. Suture the skin with catgut, and put the patient to bed upon his back. As after any operation on bone, if the wound be not sewn up too exactly, seepage of blood through it will be facilitated, and the formation of a hematoma will be discouraged. The wound is dressed after a week or ten days. The patient lies flat on his back for six weeks. Hibbs applies a specially made Taylor brace immediately after the operation. Children do well on a Whitman-Bradford frame.

For six months after the operation the patient wears a Taylor brace, or a plaster jacket. By the end of this time, firm bony fusion of the laminae should have occurred, though Hibbs advises splinting for about a year. Laboratory specimens and secondary operations show that this fusion actually does take place. Secondary operations show that persistence of pain, and further progress of the disease may be due to unskilful operating. In spite of apparently unfavorable conditions for wound healing,

experience shows that infection of the wound is very rare, and usually is of little moment when it does occur.

The Bradford frame is made of gas pipe, covered with canvas, in three pieces, of which the middle one under the



FIG. 81.—Antero-posterior view of the preceding. Note the old calcified psoas abscess.

buttocks is removable to facilitate the introduction of the bed pan. This frame is slightly wider than the patient's body. It rests on the bed, and the patient is bound to it with a swathe.

Whitman's modification of Bradford's frame is preferred by many, as affording better immobilization. Its

frame also is of gas pipe, usually of a calibre of three-eighths to a half inch, with ordinary elbows at the four corners. It is provided with a canvas cover, laced up the back, and with straps at the top and bottom, so that it can be drawn very tight. A canvas apron with straps attached for insertion into buckles sewn to the canvas cover, serves to secure the patient to the frame. This frame is much narrower than the original frame of Bradford, having a width equal to the distance between the patient's shoulder joints, and a length about eight inches greater than his height. It is very convenient for carrying the patient about, and affords perhaps better immobilization for the spine than any other form of apparatus. A piece of rubber sheeting sewn to the canvas cover under the buttocks prevents soiling. It can be easily manufactured by a plumber and harness maker.

The Taylor brace has a framework of two steel upright bars attached below to a thin, flat, steel pelvic band, about an inch and a half or two inches wide. The steel uprights run parallel, about an inch and a half to two inches apart, according to the patient's size, flare slightly outward above, and extend to a point just below the upper lateral border of the trapezius muscles. They are fitted accurately to the curves of the spine, rest upon the lateral masses of the vertebræ, and are provided with padding on the side toward the body. The pelvic band about half encircles the body between the femoral trochanters and the crests of the ilia, and it also is lightly padded with leather on its inner aspect. The frame is held to the body as a splint, above by means of shoulder straps attaching to the steel uprights at their top and at their middle, below by an abdominal apron buckling by straps to the pelvic band and to the uprights.

Instead of any of these forms of apparatus a plaster jacket may be employed, but great care must be exercised

to prevent excoriations over the fresh wound. A plaster bed does not entail so much danger of these. This should be made a few days before the operation. With the patient lying on his face plaster bandages are passed back and forth over his trunk, and if necessary over the back of his head, and are molded snugly upon him like a shell. This shell is put away in a hot, dry place to promote setting, and receives the patient after the operation.

Occasionally persistence of pain after the lapse of time sufficient for bony union warns of its failure, perhaps between two vertebræ only. A second operation will correct this, but this occurrence becomes less frequent as skill increases.

THE ALBEE OPERATION.—Prepare the back and the right shin for operation. Having ascertained as accurately as possible the extent of the disease, make a curved incision long enough to permit operating upon the spines of the affected vertebræ and of those of the healthy vertebræ above and below them—at least of one above and one below. The incision starts at about the line of the spines, curves laterally for about an inch or more, runs parallel to the vertebral column, and curves back again to the line of the spines. Dissect the flap back and reflect it. With a very broad, very sharp Albee osteotome, chisel deeply into the spines, slightly to one side of their middle, and pry the smaller piece laterally with the osteotome. The osteotome divides not only the spines but also the interspinous ligaments, and leaves a deep wound with the raw fractured surfaces of the spines at intervals on its walls. Measure with a probe the length of this wound, and bend the probe at a point to register the required length of the graft. Cover the wound with a towel.

With the right leg flexed at the knee, make a slightly curved incision about an inch longer than the length of the proposed graft, over the antero-medial cortex of the tibia, and reflect the flap. Reflect the flap from the crest of the tibia rather than towards it. In other words make the incision convex towards the crest. Make two straight parallel incisions, about five millimetres apart, through the periosteum, whose length is that of the proposed graft, and connect their upper and lower extremities. In other words trace out the graft in the periosteum.

With a very narrow osteotome, chisel through the tibial cortex transversely at the upper and lower ends of the graft. With a motor saw,¹⁰ single or twin, saw through the tibial cortex in the line of the incision of the periosteum, having an assistant pour water on the saw meanwhile to keep it from getting hot. Remove the graft and grasp it with two pairs of Kocher forceps. Turn the wound in the leg over to an assistant for closure, or cover it with a towel for closure when the rest of the work is done. Wedge the



FIG. 82.—Specimen removed at necropsy from a case of hepatic cirrhosis. An Albee spine operation had been done for spinal tuberculosis two years previously, and a cure apparently had resulted. The graft is on the left. It had solidly united to the spinous processes. A section was removed at A for microscopical study.

¹⁰ In case of need the graft can be removed with mallet and osteotome, but the operation is much more difficult, and the pain after the operation is wont to be greater.

graft between the divided spinous processes, and suture it firmly in place with sutures of kangaroo tendon, or chromicised catgut through the interspinous ligaments. Close the wound. Put the patient to bed on his back, and keep him there for six weeks. The wound may be dressed in a week or ten days. Prudence dictates the application of a plaster jacket, or a brace for several months thereafter.

In favorable cases, new bone is laid down upon the trabeculae of the graft, and the spinous processes become united with a firm bony bridge. Occasionally the graft is cast out, and the operation is a failure.

In order to avoid the wound in the shin, some operators have made use of boiled bone from other animals, but the consensus of opinion seems to be that these heteroplastic grafts are inferior to the so-called autoplastic. The greater ease with which boiled bone is absorbed throws the scale against success in a certain proportion of cases.

NON-OPERATIVE TREATMENT

This can be carried out in recumbency on a frame, or with some form of ambulatory apparatus, such as the Taylor brace or a plaster of Paris jacket. Leather jackets, moulded on a plaster cast of the trunk, steel and celluloid corsets, etc., have their advocates.

The frame is especially adapted to young children, and to meet special indications in older children, such as the onset of a paraplegia, or the occurrence of abscess. To adults, whose spines have fixed curves, the frame is not so well adapted. The Whitman-Bradford frame, when bent backward at the seat of deformity, tends to reduce the kyphosis. In the past the forcible correction of the deformity, under an anæsthetic, has been advocated on the theory that nature would build new bone in the gap left

by the separation of the vertebral bodies, but this she seems unable to do. The operation is not without danger, and has been generally abandoned.

The plaster of Paris jacket forms an excellent splint for routine employment, and should be put on with the patient partly suspended. The ordinary jacket is of little



FIG. 83.—Ordinary plaster jacket. In the spine of the patient on the left the disease is rather high up, and the jacket evidently is not controlling the deformity.

use in disease above the seventh cervical vertebra and it must be made as long as possible, consistent with the use of the limbs. The line of its upper border runs from just below the upper border of the sternum, curves sharply downward at about the nipple line, under the axillæ, and so across the back. The lower border reaches almost to the pubes, then curves upward to allow flexion of the thighs.

For disease of the upper thoracic or of the cervical spine the base of a so-called jury mast can be incorporated in the plaster jacket, and from this jury mast the head of the child may be slung. It is rather an unsightly, and by no means a satisfactory appliance, and has been more or less superseded by the "grand" Calot jacket, which provides support for the chin and occiput. This jacket is not

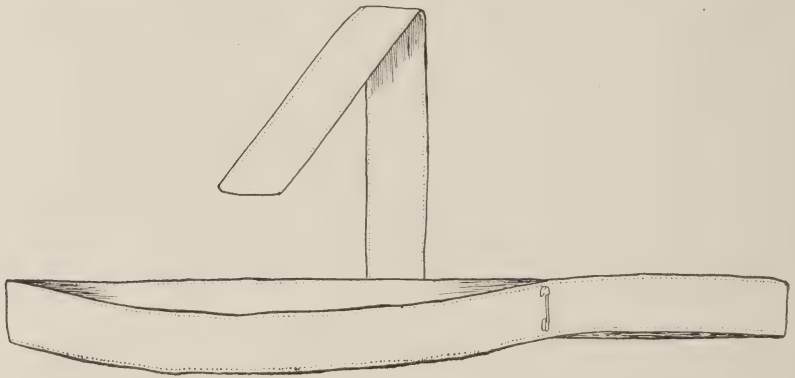


FIG. 84.—Calot head sling. One safety pin has been inserted.

easy to make, and the surgeon who has never applied one will do well to practise on the mannikin.

The Calot head sling is an improvement over the old fashioned kind, and is made of strips of canvas 6 centimetres wide. To a circular piece 168 centimetres in circumference, is sewn a tail piece 104 centimetres long, at right angles to it. With the tail piece exactly in the middle the circular piece is laid flat upon the table. This gives the contrivance a T-shape, with the doubled circular strip for the cross piece. If the patient's occipitofrontal circumference be taken, if two centimetres be added to this, if one quarter of this distance be measured off on each side from the middle of the tail piece along the circular strip, and if the circular strip be fastened together with safety

pins at these points, a sling results which can be just slipped over the patient's head under his chin and occiput. The part of the circular strip on the outside of the safety pin passes up at the side of the patient's head, and with its fellow on the other side serves as a sling to suspend the patient from a cross bar. The tail piece starts from the child's occiput, and passes up to be attached to the middle of the transverse bar. It keeps the patient's head from tilting backward, when his heels are pulled clear of the floor.

The patient wears two shirts of stockinette, the inner one provided with short sleeves, and long enough to be pinned over the top of the head during the application of the jacket, the outer one sleeveless, and reaching to the neck. A nose hole is cut in the inner one to allow free breathing. The chin, occiput, pelvis, and the kyphosis are then well padded, and a large triangular pad of cotton wadding, or of cotton batting, about an inch thick, is held in place by an assistant, over the sternum and the anterior aspect of the ribs. Another assistant holds the patient's arms at an angle of about 45 degrees to his body. Two or three plaster bandages are then applied rapidly and smoothly to the head, neck and trunk of the patient, leaving the breathing hole, of course, free. An assistant slits the borders of these bandages with scissors where they tend to draw. Then the auxiliary pieces, which an assistant has been preparing, are applied, and over these several more plaster bandages—enough to give the jacket the required strength.

While the plaster is drying, the surgeon and his assistants give their attention to molding it as accurately as possible upon the patient, especially about the shoulders and pelvis. Through a small triangular opening cut over the sternum the large pad of cotton is removed, and the jacket

is trimmed off above, below, and at the junction of the arms with the body. The line of the jacket above runs



FIG. 85.—The "grand" Calot jacket.

from just below the mouth, curves gently downward to clear the ears, and then rather more sharply upward again to include the occiput. The patient is removed from the sling, and is laid face downward, with his head over the edge of the table. A nurse should watch his breathing for a while to see that it is not impeded.

About the end of 48 hours, the jacket should be cut out in front, as shown in the illustration. If the surgeon wishes to attempt to reduce the deformity, he may also cut a window 3 x 6 inches over the kyphosis, and, having slit the shirt and greased the skin, tuck between them oblong pieces of cotton wadding, somewhat larger than the window, by means of a spatula. The shirt may then be folded back into place, and the whole thing held in place by a plaster band-

age. This procedure tends to drive the kyphosis forward, and may be repeated at intervals of two months. A jacket should last for about six months.

The auxiliary pieces mentioned above consist of three or four layers of crinoline impregnated with fresh plaster

cream. They are four in number, two aprons, a chin piece, and a piece for the occiput. The aprons are one and a half times the length of the trunk, and in width are equal to one-half its circumference. The anterior one is applied from the top of the sternum to the pubes, and its redundant portion is folded up over the lower abdomen. The posterior one is slit up for almost half of its length, making two tails, and these tails are brought over the shoulders, and then down in front of the chest, and each into its own axilla. As with the plaster bandages an assistant stands ready to nick the aprons with scissors, to make them lie smoothly.

The pieces for the chin and occiput, about 4 x 6 inches in diameter, are applied to the front and back of the neck, and reinforce the jacket in these regions.

POTT'S PARAPLEGIA.—This is best treated by recumbency, reinforced by a frame or perhaps by a Calot jacket. The outcome is usually good. The operation of laminectomy has been recommended by some surgeons. Menard advised costotransversectomy to remove the broken down material on the outside of the dura.¹¹



FIG. 86.—The "grand" Calot jacket, side view.

¹¹ GOLDMANN: "Ueber die chirurgische Behandlung der Spondylitis tuberculosa." *Münch med. Woch.*, 1909, lvi, 2341.

TREATMENT OF COLD ABSCESS.—The secret of the treatment of a cold abscess is to attack it before it grows too large, and before it approaches the surface too closely. The postpharyngeal abscess may be aspirated from the side of the neck, and if the surgeon wishes, he may inject its cavity with an iodoform mixture, or with any sterile mixture he fancies. Psoas abscesses should be attacked before they pass under Poupart's ligament into the thigh. A large aspirating needle is thrust inwards and upwards at a point one centimetre distal and two centimetres medial to the anterior superior spine. The dressing should be so bandaged on as to exert continuous pressure thereafter, and the aspiration should be repeated as often as the abscess fills. One's efforts in this line will be more successful if the patient be put to bed. Otherwise the force of gravity must be combated.

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CHAPTER II

TUBERCULOSIS OF THE HIP

WHILE presumably the disease may occasionally start in the synovial membrane, the original focus in the great majority of cases is probably in the bone. It may be in the acetabulum, or more often in the head or neck of the femur. If the head and neck of many femora be sawn into thin slices, a small area will usually be found in the distal and medial portion of the head, near the joint surface, in which the bone trabeculae are very scant, and in which there is little else but marrow.¹ This seems to be the favorite starting place of the disease.

In rare instances the marrow of the great trochanter may be attacked, and the hip joint itself may never be invaded at all. As a rule the disease does not travel widely in the os innominatum unless secondary infection take place, but remains limited to the region of the acetabulum. The tuberculous granulations may make their way through this, perforate it so to speak, and give rise to an intrapelvic abscess. The customary place of exit of a cold abscess from the joint is at its distal and medial aspect, where the capsule is weak.

As a result of the pressure of the head of the adducted femur on its proximal rim, more or less softened by disease, the proximal portion of the acetabulum undergoes partial absorption, and the head of the femur becomes subluxated proximally, though practically never does actual luxation take place. The acetabulum becomes elongated, shaped

¹ ELY, LEONARD W.: "A study of 100 dry bones sawn in the laboratory: Bone and joint studies, I." Published by Leland Stanford University, 1916.

somewhat like an oyster shell. This is the so-called "wan-dering acetabulum" of hip joint disease.

SYMPTOMATOLOGY

The pain in hip joint disease may be referred to the joint itself, or, especially in children, to the medial aspect of the knee. In the early stages of the disease, it may be worse in the morning, when the patient begins to go about, growing better on exercise, but generally it is made worse by motion of the joint, as is usual in tuberculous joint disease. It is more or less constant, though it may come on in spasms at night, and may cause the notorious night cries, described in the preceding section. It may cease entirely if the joint be immobilized.

To the pain is also added a feeling of stiffness, and in adults the stiffness may be more prominent than the actual pain, and, like it, may be quite marked in the morning, wearing off as the limb is used, that is, in the early stages of the trouble. The stiffness and the pain cause a limp. Muscular spasm is a constant physical sign. The patient often complains that one of his legs is shorter than the other, usually the affected one.

When the patient is stripped, and is laid supine upon a firm examining table, a change of attitude may be noted, sometimes, in the early stages or after the rupture of an intraarticular abscess, an attitude of slight abduction and external rotation. The characteristic attitude of hip joint disease however is one of flexion, adduction and internal rotation. The recognition of the attitude of the limb, and its interpretation is sometimes difficult to the uninitiated, and a few words upon the symptom-complex may be in order.

When the normal child lies supine, his lumbar spine,

his hips, and his lower extremities rest upon the table, and if his heels are together, his extremities will roll somewhat outward, and his feet will be seen slightly to diverge. In an adult the lumbar spine is close to the table if it does not touch it. This is the normal posture of extension, and of slight external rotation, indifferent as to abduction or adduction. Any deviation from normal as to rotation can be distinguished easily by comparison with the well side.

If fixed flexion be present, the popliteal space leaves the table, and the thigh is seen to be at an angle with the trunk; but it must be remembered that the normal lumbar spine is flexible, and, if it arches upward, the knee can be crowded down upon the table. This will disguise the presence of any ordinary degree of flexion in the hip. Hence in determining the presence of fixed flexion one must be sure that the lumbar spine is flat upon the table. It may be held there by flexion of the sound hip. If fixed flexion be actually present upon the affected side, the knee on that side cannot then be brought down upon the table.

The attitudes of fixed abduction and fixed adduction, are not so easily understood, but a little study clears them up. Normally in standing or lying, the thighs are neither in abduction nor in adduction, but are parallel (Fig. 88 1, c, d). Now if we suppose that the left thigh is adducted (Fig. 88: 2,d), and that the pelvis (b), and the spine (a), are fixed and immovable, then the left thigh (d) will be crossed over the right thigh, and progression would be extremely difficult; but the lumbar spine is flexible, and the patient, in order to bring the thighs into line, tilts the pelvis up on the affected side (Fig. 87; 31). At the same time, it is seen that this manœuvre throws the sound side into abduction, and draws up the affected extremity, so

that it looks short—*apparent shortening*—while measurement with the tape shows that the two extremities are of the same length.

An analogous state of affairs is present when the limb is in abduction (Fig. 88; 4 and 5). Adduction makes apparent shortening, abduction makes apparent lengthening.

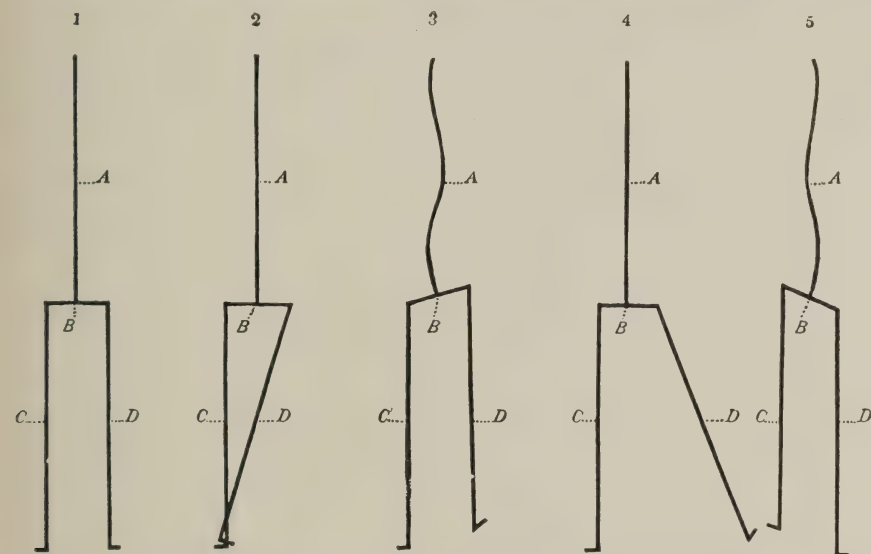


FIG. 87.—Diagrams showing the effect of fixed adduction and of fixed abduction upon the attitude of the lower extremity.

The change in the attitude of the limb is responsible for various physical signs that are often described; *e.g.*, if the thigh is in flexion, the gluteal fold will be shorter and shallower, and the fold of the groin deeper than on the sound side. If the limb is in adduction the fold of the groin is deepened, if in abduction it is made shallower.

The trochanter is at Nélaton's line, or in old cases with wandering acetabulum slightly above it.

Limitation of motion will usually be present in all directions, especially of abduction, and it varies from a

slight restriction at the extremes to a practically complete abolition of motion. Attempts at passive motion cause pain and muscular spasm.

To elicit the limitation of motion one puts the joint through its passive range in every direction, comparing each motion with that on the sound side. To ask the patient to perform the motions actively is to introduce an element of confusion, and subserves no useful purpose. It is well to adopt a regular routine, and to follow it methodically. With the knee of the affected limb held to the table, one flexes the sound hip, with the knee flexed, and notes its range. One repeats the manœuvre with the affected hip, and compares the result. Then with the hip and knee in right angle flexion one tests the range of abduction and of adduction, first in the sound hip, and then in the affected one. In the same attitude, and in the same way one tests the range of internal and of external rotation. Finally, one turns the patient on his face, and tests the range of extension in each hip, holding the other one flat on the table.

Sensitiveness and slight swelling can sometimes be detected over the head of the bone, and occasionally, thickening about the trochanter. The practice of striking the soles of the feet in an attempt to elicit pain is unnecessary, and, as it inflicts trauma, is unwise.

Atrophy of the thigh and of the calf often can be discerned by the naked eye, but should be accurately determined with the aid of a tape, measuring at corresponding levels on the two sides, that is, at a certain distance from the patella. Generally speaking, the atrophy of a tuberculous arthritis is greater than that of other forms of arthritis of an equal severity.

It is customary to determine the actual shortening or lengthening by measuring from the anterior superior spine to the tip of the medial malleolus, and, while this measurement is not accurate, it is sufficient for all practical purposes. In the early stages of the disease a slight increase of length can sometimes be detected, probably the result of stimulation of growth caused by the inflammatory process close to the epiphysial disc. Thereafter growth lags behind, and measurement shows an actual shortening. This shortening is rarely great, usually about a centimetre or two, but it persists through life. It is due to three things: 1st. Interference with growth at the epiphyseal disc, 2nd. Destruction of much or little of the head of the femur, 3rd. Subluxation of the head proximally in the wandering acetabulum. Parenthetically it may be remarked that the left lower extremity is usually longer than the right.

Measurement from the umbilicus to the medial malleoli shows the apparent shortening or the apparent lengthening. As far as the patient himself is concerned, the apparent lengthening or shortening is the practical lengthening or shortening; for if the extremity be drawn up, it is functionally short as well as apparently so, and the patient will always complain that his leg is short.

DIFFERENTIAL DIAGNOSIS

What already has been said in the section on the general differential diagnosis, applies to the hip. Several lesions however deserve special mention.

IN CONGENITAL DISLOCATION the limp will have been present from infancy, that is, from the time the child began to walk. The joint will not be painful, muscular spasm and signs of inflammation will be absent, abduction alone will

be limited, the shortening will amount to an inch or more, and finally, the head of the bone will be found both by palpation and by the Röntgen rays, to be out of its socket.

LEGG'S DISEASE, the so-called Perthe's disease, "*arthritis deformans juvenilis*," gives a clinical picture very like that of tuberculosis. Only during the last few years have the two been differentiated. It is probably the recovery of patients with Legg's disease, that has made certain surgeons optimistic in their view of hip joint tuberculosis. In Legg's disease, the patient, usually a boy between five and ten years of age, complains of a painless limp. The hip is usually in slight external rotation, and, when it is flexed, goes into slight abduction. Muscular spasm is very slight, if present at all. Abduction is greatly limited, other motions may be limited or free. The diagnosis is made with the aid of the X-rays. In Legg's disease, most of the change takes place in the epiphysis itself, which becomes flattened, segmented, and often displaced laterally. Areas of rarefaction may or may not appear in the neck, which becomes shortened, thickened, and often bent at a more acute angle with the shaft—*coxa vara*.

COXA VARA (bending of the femoral neck) is a disease, or rather a deformity of adolescence, causing limp, limitation of motion, stiffness and some pain. Its cause in the great majority of cases is probably an old unrecognized Legg's disease, or an old healed fracture of the femoral neck. In localities where joint conditions receive proper attention this condition is not seen as often as formerly. The limb is in an attitude of adduction, extension and outward rotation, while abduction, flexion and internal rotation are limited. Actual shortening of one to two centimetres is present, the trochanter is prominent, and the X-rays show the head of the femoral head in the socket,

and the neck at a more acute angle with the shaft than normal.

PSOAS CONTRACTION is sometimes mistaken for hip joint disease, but with psoas contraction the hip is not adducted nor rotated inward, and when the psoas-iliacus muscle is relaxed by flexion of the thigh, all hip motions except possibly internal rotation are unrestricted. Again, an examination of the spine will often reveal the evidences of Pott's disease with psoas contraction.

PROGNOSIS

This is *quoad vitam*, good. Secondary infection with extensive suppuration in the os innominatum puts the issue in doubt. As to function authorities differ. Some surgeons believe in their ability to cure the disease by conservative treatment with excellent function, others, of which the author is one, think that a stiff hip or a loose one with an amputated femoral head is the best that can be attained.²

TREATMENT

Here again authorities differ. Some advocate radical measures in patients of all ages, others invariably carry out conservative treatment. Practically all agree that half way operative measures, such as scraping, draining and packing are worse than useless. The trend of modern opinion is toward conservative treatment in children and radical treatment in adults. The principles on which each is carried out have been set forth in the section on joint tuberculosis in general. The special measures for carry-

² On page 133 of the author's book on joint tuberculosis, appears a Röntgen picture of a supposed case of hip joint tuberculosis cured with perfect function. In the light of modern knowledge this is evidently a case of Legg's disease.

ing out these principles in disease of the hip will be detailed here.

CONSERVATIVE TREATMENT.—In an early case, without deformity, splinting may be resorted to forthwith, otherwise the first indication is to remove the deformity. This may be done in one of several ways.

1. *Rest in bed with traction.* This is done with an ordinary Buck's extension outfit. In order that traction may be made in the line of deformity, the affected limb is flexed until the lumbar spine lies flat upon the bed, and then is supported by an inclined plane. As the spasm gives way, and as the fixed flexion lessens, the inclined plane is lowered until the limb rests upon the bed. The reduction of the deformity is made easier if a hip splint is first applied. Traction may then be made upon the lower end of the splint.

2. *Slow correction with plaster of Paris spicas.* With assistants holding the limb in the best possible position, a well padded plaster spica is applied from the nipple line to the toes. This plaster remains on for several weeks. When it is removed, the deformity will be found capable of partial reduction. Another spica is then applied in the improved position, and others follow it until the deformity has been entirely corrected. This is the method of Lorenz, somewhat tedious, but safe.

3. *Immediate reduction of the deformity under ether,* followed by the application of a plaster spica, or of a hip splint, with succeeding rest in bed. This method has its disadvantages, and, on account of the unavoidable trauma to the inflamed joint, is not without danger, but if the manipulation be carried out with strong traction, and very gently and carefully, the results are usually good. It is especially indicated where the deformity is caused by muscular spasm, rather than by adhesions in the joint.

The deformity we are compelled to fight throughout the course of a hip joint tuberculosis, is one of flexion and adduction. Therefore we get the hip into an attitude of slight abduction (about 20 degrees) and full extension, and endeavor to keep it there.

After the correction of the deformity, the conservative treatment consists in rest for the joint until nature has cured the disease, meeting meanwhile any complications that may arise, and, according to the individual preference of the surgeon for immobilization or for traction, he will rely mainly upon 1, plaster of Paris, or 2, braces.

1. The short plaster of Paris spica in the treatment of hip disease was introduced by Lorenz about 1891, and has attained rather a wide vogue. It is now the routine treatment in many hospitals and clinics. It enables the patient to use the limb in walking, and so avoids the extreme atrophy that accompanies the treatment by braces. It is also more comfortable and less troublesome than the brace. With the hip in plaster the patient can go about and even play, and his general condition will usually be better than if he be hampered by a heavy brace. On the other hand, the protection afforded to the joint by a well fitting brace, is



FIG. 88.—The short plaster of Paris spica in the treatment of hip-joint tuberculosis.

thought by some to be more thorough, and better functional results have been claimed for it. The claim has also been made that with its use abscesses are rarer.

The application of a spica is made easy by the presence of a special fracture table, but portable and stationary pelvic rests are almost as serviceable, and can be used with any common strong table. The head and shoulders can rest upon an ordinary box. Two assistants are advisable, one to hold the head and shoulders on the shoulder rest, the other to stand between the legs and hold them in position. The former is not necessary, the latter is; he gives his attention to keeping the affected hip in extension and abduction.

The stockinette shirting reaches from the nipples to the malleoli, like a long pair of drawers with one leg cut off. The bony prominences of the pelvis and of the knee should be well padded with sheet wadding, silence cloth, or some similar material. No padding is necessary or advisable for the thigh muscles. A snug bandage holds the padding in place.

The plaster bandages are then applied, reversed repeatedly and piled up over the pubes, so that the plaster there will be about an inch thick, while at the knee its thickness will not amount to more than about one-eighth of an inch. While it is being applied it is thoroughly rubbed and molded, especially about the pelvis, and then it is trimmed with a sharp knife in the following manner: Above, at the side, the line starts about two inches above the iliac crest, and sweeps down across the belly with concavity upward, so as to lay bare the umbilicus, and then rises to the former level on the other side, and so straight across the back to the starting point.

Below at the knee the plaster holds the condyles and the patella, and is cut with a concavity behind so as to permit

flexion of the knee. Finally a third cut starts from below the level of the trochanter on the well side, and, arching across just at the pubes, passes two or three inches below the perineum, and then under, upward and across, clearing the anus by two or three inches, to the starting point.

After the superfluous padding has been trimmed away with bandage scissors or Lorenz shears, the shirting is turned up over the plaster and sewn together, so as to make a covering as well as a lining. "Scratch" bandages under the shirting will keep the skin in condition.

If one desire to relieve the hip of weight bearing one includes the leg in the plaster, and in the lower part includes the bars of some such appliance as a Lorenz stirrup. In this case the sole of the shoe on the sound side should be raised two or three inches by cork or wood to compensate for the extra length. The same effect can be obtained without the stirrup if the high shoe be worn on the sound side and the patient use crutches.

The Thomas hip splint, not to be confused with the more celebrated knee splint, is designed to afford fixation and not traction, and is therefore similar in its action to the plaster spica, though probably not so efficient, and more troublesome. It is not designed to permit weight bearing, and with it the patient wears a high shoe on the sound side, and goes on crutches. It consists essentially of a flat, longitudinal bar, reaching from the level of the nipple to the middle of the calf. This bar is carefully fitted, and passes straight down posteriorly, behind the

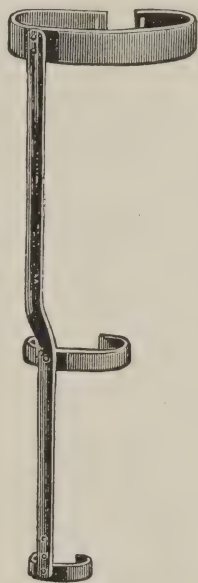


FIG. 89.—The Thomas hip splint.

tuberosity of the ischium. Three attached horizontal bands above, below, and about one-third from the bottom, partially encircle the thorax, the upper part of the thigh and the calf. This apparatus is bandaged on the patient, and a sling over the shoulders from the top cross piece, keeps it from sliding down. It is quite popular in England, but is rarely used in America.

The traction brace consists essentially of a leather-covered steel pelvic band which partly encircles the pelvis just above the trochanters, and of a steel stem running downward from the lateral aspect of the pelvic band, and at a slight angle with it corresponding to the inclination of the pelvis, to a point two and a half to three inches below the sole of the foot, where it is turned inward to form a foot plate. At the level of the knee the upright is provided with a band which encircles the knee, and steadies it.

The traction is exerted by means of long strips of mole-skin adhesive plaster on the medial and lateral aspects of the thigh and leg, provided at their lower extremities with buckles. To the foot piece of the brace are attached straps which buckle on these. Counter-extension is furnished by perineal bands, which buckle in front and behind to the pelvic band, in front directly over the origin of the adductor muscles; behind, somewhat further lateral, so that the bands will pass over the tuberculosities of the ischia. The foot piece is usually shod with rubber or leather to prevent slipping. The perineal bands may be made of stout webbing covered with two or three layers of Canton flannel to prevent chafing.

Many modifications of this brace have been devised. Sometimes the pelvic band is jointed to the upright, sometimes it is bolted fast. The upright may be provided with a rack and pinion for extension, perineal bands may be

replaced by a padded ring (Thomas ring) about the proximal end of the thigh, or there may be but one perineal band instead of two. Sometimes the pelvic band is rudimentary, sometimes the upright is provided with a lateral traction strap designed to pull the femoral head away from the socket.

The brace sometimes known as the Children's Hospital brace, of Boston has no pelvic band nor perineal straps, but instead an incomplete ring about the top of the posterior portion of the thigh, continued by a sort of U-shaped piece over the pubes on to the perineum of the sound side.

With any form of traction brace the sole of the shoe on the well side should be raised two to three inches to compensate for the length of the brace.

It is seen that none of these braces immobilizes the hip joint, and the whole theory of the traction brace rested on the idea of "motion without friction." Its advocates believed that immobilization promoted ankylosis, and that a certain degree of motion was beneficial. This theory was later somewhat modified, and motion was regarded, while not actually beneficial, at least, as harmless.

Attempts have been made to devise apparatus which should combine traction with immobilization, but the apparatus was so cumbrous that it never attained wide use.

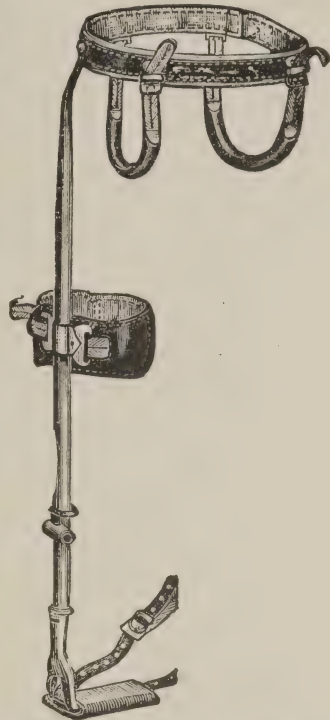


FIG. 90.—Ordinary old fashioned hip splint.

If, in spite of our efforts, cure finally takes place with the hip in a faulty attitude of flexion and adduction, no attempt should be made to correct the deformity by force. To tear loose the dense fibrous adhesions which hold the bones fast is, in the hip as in other joints, to run the risk of lighting up the disease afresh. In this fibrous tissue are locked up collections of tuberculous material. To tear it apart is to break down the barriers which nature has erected. For the same reason any mobilizing operation whatever is to be avoided. It is true that successful arthroplastic operations have been done on old, supposedly tuberculous joints, but it is also true that they have lighted up disease in old tuberculous joints, long quiescent.

Probably the safest and most satisfactory operation in these old flexed, adducted hips is an osteotomy. It may be done in one of several ways. Perhaps the best two methods are those by cuneiform osteotomy at the lesser trochanter, and by linear osteotomy through the femoral neck. If a linear osteotomy be done at the level of the lesser trochanter, the proximal end of the distal fragment is likely to be levered medially when the deformity is corrected.

CUNEIFORM OSTEOTOMY.—The operator, having gotten his bearings on the location of the lesser trochanter, by measuring on the X-ray plate, makes his longitudinal incision, about 10 centimetres long, over the postero-lateral aspect of the femur, through the periosteum, down to the bone. His assistant retracts the periosteum, and with an osteotome he removes a wedge of bone with its apex at the lesser trochanter, and its rather broad base at the postero-lateral femoral cortex. The apex does not include the medial cortex. It runs down just to it. The distal plane of the wedge is exactly transverse to the shaft, the proximal oblique. The surgeon, by forced abduction and extension,

fractures the medial cortex, corrects the deformity, closes the wound, dresses it, and applies a plaster spica from nipples to toes. At the end of about five weeks, the leg part of the spica, and about four or five inches of the top, may be removed, and the patient may begin to walk shortly thereafter. If the limb be put up in an attitude of moderate overabduction, the actual shortening will be compensated, and the patient will walk with a barely perceptible limp. The result is a satisfactory one; the disadvantage is the unsightly anterior protrusion of the proximal fragment. In a woman it is well to err on the side of overabduction.

A good incision for osteotomy of the neck is a straight one 15 centimetres long, from the anterior superior spine distally between the tensor fasciæ femoris and the sartorius. It is carried down to the neck, the tissues are retracted, and the neck is divided with mallet and osteotome. Dressings and spica are applied as in the preceding operation. Some authorities discountenance this operation on account of the risk of lighting up the disease,³ but if bony union ensue, the danger is probably very slight. A fracture table makes this operation and the preceding quite simple.

OPERATIVE TREATMENT

Unless the surgeon cling to the old and discredited idea of scraping out and draining a tuberculous joint, or unless he throw up the sponge and amputate, he will approach the problem with one of three ideas in his mind. He will endeavor (*a*) to remove all the tuberculous tissue, (*b*) to ankylose, or (*c*) to resect and (1) dislocate, or (2) shove the trochanter into the acetabulum.

³ VINCENT, EUGENE: "Osteotomie et ostectomie pour ankylosis viciuses de la hanche." *Revue de Chir.*, 1902, ii, 465.

(a.) The practical impossibility of removing all diseased tissue from a tuberculous joint already has been explained, and yet operations done with this purpose often are successful, not because the theory on which they are done is correct, but because they accomplish a result which was not in the operator's mind. They vary from a simple "tunneling" operation through the lateral cortex into the neck,^{4, 5, 6} to a wide resection of the head, neck, greater trochanter and acetabulum. Every effort is made to follow the disease with chisel, gouge, knife and scissors, and then usually the stump of the femur is thrust into what remains of the acetabulum, the wound is closed and dressed, and a plaster spica is applied with the limb in abduction.

(b.) The hip is not an easy joint to ankylose, and, before attempting to ankylose it, the surgeon should decide whether the patient's mode of life will be best subserved by a perfectly stiff hip, or a loose but fairly stable one. If the former, the Albee operation perhaps is the best, if the latter, then the hip should be resected. Most operators prefer the resection.

THE ALBEE OPERATION.⁷—The ordinary anterior incision, 15 centimetres long, distal from the anterior superior spine between the sartorius and the tensor fascial latae, is carried down to the capsule, and the capsule is opened. With mallet and osteotome, a slice is removed from the proximal portion of the acetabulum, and a corresponding slice from the proximal portion of the femoral

⁴ MACNAMARA, CHARLES: "Diseases of the bones." London, J. and A. Churchill 1881.

⁵ HUNTINGTON, T.: "The early operative treatment of osteomyelitis in the femoral head and neck." *Surg., Gyn. Obst.*, 1906, ii, 409.

⁶ SHERMAN, H.: "Report of focal operations in hip joint tuberculosis." *Cal. S. J. Med.*, 1907, v, 62.

⁷ ALBEE, T. H.: *Surg., Gyn. and Obst.*, 1910 x, 256.

head. This permits a slight **subluxation** proximally, of the head of the femur. As much as possible of the cartilage is removed from the femoral head together with a little of the bone underlying it. An assistant rotates the femur in and out, to facilitate this. The wound is closed with superficial and deep sutures, and dressed. A long plaster spica is applied, with the hip in slight flexion and abduction.

RESECTION.—A simple resection of the femoral head in an uninfected case of hip tuberculosis can usually be relied on for a cure, whether the operator lets the great trochanter dislocate on the dorsum of the ilium, or shoves it into the acetabulum. The latter operation is not so apt to be followed by extreme and troublesome adduction, as the former. We do not know the exact reason why these simple operations, without any pretense of removing all the tuberculous tissue, are a success, and we shall not know until we have had the chance to examine specimens from cured cases in the laboratory. Probably the same change of the lymphoid marrow to fatty as follows an ankylosis, takes place here and the bone becomes dense. Dense bone is a poor location for tuberculosis. The disease seems to require some space to thrive. Any one of a number of incisions may be employed for this operation.⁸ The wound should always be closed without drainage, and the hip should be put up in a long spica in rather marked abduction. The plaster may be trimmed for 5 or 6 inches above, and at the knee, in about a month, and the patient may walk shortly thereafter.

The anterior incision has already been described. It does not afford a wide view of the joint, and, if much

⁸ BRACKET, E. G.: "A study of the different approaches to the hip joint, with special reference to the operations for curved osteotomy and for arthrodesis." *Boston Med. and Surg. Jour.*, 1912, clxvi, 235.

obstruction to removing the head of the bone exists, it is not very satisfactory. The operator who employs it should take care to keep lateral to the sartorius muscle. If he carry his dissection medial to it he is likely to have trouble with branches of the femoral nerve. It is an excellent incision for a simple case.

SPRENGEL'S INCISION.⁹—This was brought out many years ago, and has recently been revived under the name of Smith-Peterson. It is very simple, very satisfactory for all ordinary cases, and has not attained the popularity it deserves. It has two arms. The longitudinal one starts from the anterior superior spine of the ilium, and runs distal about 15 centimetres just lateral to the tensor fasciæ latæ. The transverse arm runs from the anterior superior spine backward along the outer lip of the crest of the ilium for about half its length, and is carried down right through the periosteum to the bone.

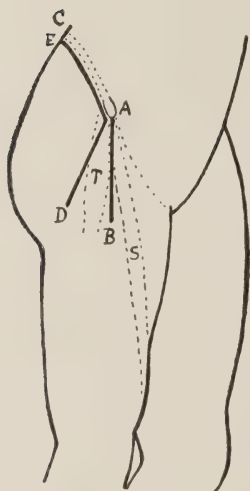


FIG. 91.—Incisions for hip joint resection. A, B, ordinary anterior incision. E, A, B, Smith-Peterson incision. E, A, D, Sprengel incision. S, sartorius muscle. T, tensor fasciæ femoris. The dotted lines C, A, shows the crest of the ilium.

Keeping close to the bone, the operator then pushes with a blunt chisel the fibres of the glutei, medius and minimus, away from their attachment to the dorsum of the ilium, and reflects backward the whole flap of skin, fascia, fat and muscle, until he reaches the antero-proximal border of the acetabulum. Meantime he has been carrying the longitudinal arm of the incision by intermuscular dissection down to the capsule of the joint. He opens the capsule and an assistant by exter-

⁹ SPRENGEL: "Zur operativen Nachbehandlung alter Hüftresektionen." *Archiv. f. klin. Chir.*, 1898, lvii, 837.

nal rotation turns the head out of its socket. It is best to remove it at the very base of the neck. If there be any difficulty in disarticulating, the removal of a little bone from the antero-superior border of the acetabulum overcomes it. My experience with this incision has been very satisfactory, and I recommend it for routine work.

After removal of the head, the trochanter may be permitted to dislocate, or it may be shoved into the acetabulum. The wound is closed and dressed, and the whole limb is put up in a long plaster spica, in a position of extreme abduction.

KOCHER'S INCISION.¹⁰—Place the patient on his sound side in the latero-ventral position. Slightly flex the hip. Let an assistant grasp the leg so as to change the position of the thigh according to directions.

Beginning at the posterior margin of the base of the trochanter major, make a cut in the proximal direction to the posterior angle of the summit of the trochanter. At this point change the direction of the incision, and cut proximally and backward towards the posterior superior iliac spine, *i.e.*, cut parallel to the fibres of the *gluteus maximus* and expose that muscle. Split the tendon of the *gluteus maximus* in the direction of its fibres, and enlarge the deep wound proximally and backward by splitting the muscle itself. Retract the edges of the deep wound, exposing the *gluteus medius* at its insertion into the trochanter.

Rotate the hip slightly inward, so as to make prominent the posterior part of the summit of the trochanter. Find the groove between the *gluteus medius* and *minimus*, proximally, and the *pyriformis*, distally. Beginning at this place separate with elevator or knife the insertions of the *gluteus medius* and *minimus*, along with the corresponding

¹⁰ From Binnie's Operative Surgery.

periosteum, from the trochanter, until the anterior intertrochanteric line is reached. At this point separate the insertion of the iliofemoral ligament. While doing this flex the thigh and rotate it out.

Divide the capsule along the distal edge of the pyramiformis tendon. Flex the thigh and rotate it inwards so

as to gain access to and divide the insertion of the pyramiformis. With elevator or chisel (removing a thin shell of bone if desired) separate the insertions of the obturators and the gemelli.

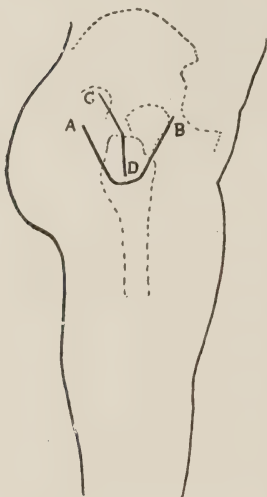


FIG. 92.—The Kocher and the Murphy incisions for resection of the hip. A, B, Murphy. C, D, Kocher.

Murphy¹¹ employed a lateral U-shaped incision, with the trochanter in its centre, reaching from a point ten centimetres proximal to one five centimetres distal to the tip of the trochanter. The open end of the U is proximal and is twelve centimetres wide. He reflected proximally the U-shaped flap of skin, superficial fascia and fascia lata. He then passed a Gigli saw about the base of the trochanter, and divided the trochanter. (This may be done with osteotome.) The trochanter, with its attached muscles, he turned proximally, and thus gained access to the capsule.

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¹¹ MURPHY, JOHN B.: *Journal of A. M. A.*, 1905.

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CHAPTER III

TUBERCULOSIS OF THE KNEE

THE primary focus may be in the tibia, the femur, or the patella. Perhaps in rare instances, the disease may start in the head of the fibula. Most authorities agree that primary synovial tuberculosis is fairly frequent, and, while this theory is practically impossible of demonstration, I believe that it is correct, especially in adults. Sometimes the location of the primary focus can be detected with reasonable certainty, often it can only be surmised.

The knee joint may be considered as comprising three articulations, two tibio-femoral, and one femoro-patellar, and this more or less complex arrangement, coupled with the presence of the synovial curtains, or partitions, is often responsible, in knee joint tuberculosis, for a peculiar distribution of the disease which has not received the attention it deserves. Thus one finds, for instance, cases with marked involvement of the quadriceps pouch, and with the tibio-femoral articulation practically intact, as if it had been walled off by adhesions in the same way as is the peritoneum. Again one finds one tibio-condylar articulation badly diseased, and the other only slightly affected. Often of course the whole knee joint is equally involved.

The joint cavity may be filled with a clear serous, a hemorrhagic, a turbid, or a flocculent fluid, or it may contain the so-called tuberculous pus. In the last case, the joint cavity constitutes a cold abscess. There is usually nothing characteristic about the appearance of the fluid in a tuberculous joint, unless it happen to be turbid and flocculent. This is practically pathognomonic. In some



FIG. 93.—Old tuberculosis of the knee-joint. The heavy shadows in the plate are not to be interpreted as new bone, but as collections of calcified material. Note the decrease in the light zone between the bones, showing the disappearance of the articular cartilages.

cases the joint contains no fluid at all. These are usually the slow, dry cases. In these the joint cavity may be absent, and the bone ends may be bound tightly together by

fibrous adhesions, which run not only from bone to bone, but also from each bone to the capsule. When these adhesions are torn apart at operation they leave the bones with an irregular, torn, disorganized appearance, that is almost diagnostic.

In some cases the synovial membrane is thickened, villous and succulent, giving a boggy, doughy feel to the articulation. These usually contain a little free fluid, not always capable of detection clinically.

In the cases with free fluid, the patella floats; in the boggy cases, it can be moved with a sort of soft resistance; in the fibrous it is usually bound tightly down to the femoral condyles.

The ends of the femur and of the tibia often appear to be enlarged, but they never are. The swelling is in the circumarticular tissues. This swelling, in the cases with much proliferation in the synovial membrane, takes on a peculiar spindle shape, accentuated by the atrophy of the thigh and calf, and the skin, with veins dilated, becomes blanched, giving to the joint an appearance from which the disease derived its former name of "tumor albus" or "white swelling." The same thing is seen sometimes in the elbow, less often in the ankle, and rarely in the other joints.

The knee is held in slight flexion, and the flexors, working at an advantage over the large extensor, sublunate the tibia backward, at the same time rotating it outward. The posterior portion of the capsule shrinks after a while, if the subluxation is permitted to remain, and prevents the return of the tibial head to its place under the condyles. If force be employed in the attempt to reduce the subluxation, the tibial head is levered backward, and possibly one

of the soft bones may be fractured. This subluxation is perhaps most prominent in the cases with marked involvement of the quadriceps pouch, and in mild synovial cases is not to be looked for.

Atrophy of the muscles of the thigh and calf is an early and constant phenomenon, especially in the bony type of the disease. In this atrophy the bones of the thigh and leg take part.

SYMPTOMATOLOGY

The disease begins with pain in the knee, stiffness and a feeling of discomfort. The patient limps. Swelling usually appears early. The swelling may be fluctuating or boggy, the patella may float or it may not. In the late stages of the disease the mobility of the patella is almost always impaired. The knee is in flexion, and its range of flexion and extension is limited. Sometimes the patient walks upon the toes of the affected foot. The flexion in the later stages may reach an extreme degree, and, with the peculiar outward rotation and general appearance of the joint, may make the identification of the disease an easy matter. In some cases however the patient may present a slightly swollen, painful joint, with nothing characteristic about it.

Sensitiveness to pressure of the synovial membrane is usually present, best detected when the knee is in flexion.

Abscess formation is fairly frequent, and, on account of the nearness of the joint to the surface, rupture of the abscess and secondary infection are hard to avoid. Luckily an infected abscess in a joint that is near to the surface is not as a rule so hard to heal as one in a deeper joint, like the spine and hip.

PROGNOSIS

There seems to be a rather definite opinion that the prognosis in knee joint disease is better than in tuberculosis of other joints, and I believe this is correct, and that it is correct because the knee is easier to stiffen. Perhaps, on the other hand, it is because mechanical conditions make splinting more effectual.

Conservative treatment requires two or three years at best, and under it children sometimes recover with a fair degree of motion, though always with the danger of a relapse threatening them, as with tuberculosis of any other organ. Some good authorities assert that the synovial form in adults often recovers with good function, but usually when good motion results, one views one's diagnosis with scepticism. At best, under conservative treatment, in the adult one looks for a painless, stiff joint, and to attain this long continued treatment is necessary. The resulting ankylosis is fibrous. Unless a secondary infection has been added, bony ankylosis is rare, if it ever occurs.

Under properly planned radical treatment, the disease can be brought to a standstill in about six months. Some patients, however, seem to possess no resistance to the disease. In them, in spite of treatment, the disease steadily advances, the operative wound breaks down, and a thigh amputation must be done to save the patient's life. We see the same thing here as in pulmonary tuberculosis, but in this case we have a life-saving measure which we do not possess in that.

TREATMENT

Some surgeons recommend conservative treatment in patients of all ages, some radical in them all, but the trend of modern treatment, in which I heartily concur, is toward

invariable conservative treatment in children, and invariable radical treatment in adults, as soon as a positive diagnosis is made. When we speak of a positive diagnosis we do not mean one that is made after a rapid examination, however experienced the examiner may be, but one founded on indisputable evidence. On the other hand, if physical examination, backed by the evidence accorded by the Röntgen rays, shows that the joint is so badly damaged as to be incapable of good function, whatever the exact nature of the disease may be, positive proof of the presence of the tubercle bacillus is unnecessary, and we proceed to complete the task which nature has set for herself, and destroy the joint.

A completely ankylosed, painless joint is better than a partially ankylosed, painful one, and far safer. There is little hope that this method of treatment will ever become very popular among the laity. The usual adult will resort to the surgeon who will promise him something better, but sooner or later he must submit to the inevitable.

In spite of the claims that have been made for conservative treatment in children, if conditions were the same in them as in adults we should probably be tempted to adopt radical measures, and spare them the long, tedious and uncertain course of conservative treatment, but in the knee, especially, radical treatment is to be shunned at all costs. Here are located two of the most important centres of bony growth in the body, and here unsightly postoperative deformity is notoriously difficult to avoid. Again, radical treatment is not followed by the almost certain cure that follows it in the adult. If my theory, whose truth has not yet been completely demonstrated, is correct, this is because lymphoid marrow is present in the shafts as well as in the ends of the bones, and its presence

is not dependent upon function. Resection does not cause its disappearance.

In spite of all that has been said for many years against the radical treatment of tuberculous knees in children, its sporadic advocacy is perennial, and evidently cannot be killed. Only the opportunity to see, years afterward, the results of their treatment, causes its advocates to abandon it, and their experience seems powerless to deter their successors.

In this connection it may be said that Hibbs maintains that his ankylosing operation does not disturb the centres of growth, and hence does not cause shortening.

CONSERVATIVE TREATMENT

Precedent to the application of permanent apparatus, any deformity in the nature of fixed flexion should be corrected, and this may require patience, care and some skill. Not only the hamstrings are contracted, but also the posterior and lateral ligaments of the joint, and, if the tibia be simply straightened by direct force, its head will be levered backward with the lateral ligaments as a sort of fulcrum, perhaps fracturing the softened bone, and the tibia will be brought into a line parallel with that of the femur, but on a plane posterior to it.

Various expedients have been devised to correct the deformity. Bilioth recommended two lateral hinges with long iron bars whose ends are provided with perforated pieces of tin for incorporation in plaster of Paris. The limb is put up in plaster piled quite thick in the popliteal space. When the plaster has set, it is divided at the knee joint, and a wooden wedge is driven in the crack posteriorly, forcing it somewhat open. From time to time other

larger wedges are driven in, until the leg is straightened. Several plaster dressings may be necessary.

The patient may be put to bed, perhaps with his *leg* in plaster, and then the leg may be put in a sling, and traction forward may be made on its proximal part by means of weight and pulley from a bar over the bed. The weight of the foot and of the distal part of the leg, tends to stretch the contracted ligaments, and to lever the tibial head out from under the femoral condyles.

No marked flexion deformity at the knee should ever be corrected too rapidly and forcibly, on account of the danger of rupturing the popliteal vessels. Gangrene has been caused by rough manipulation.

After the reduction of the deformity, the surgeon will have the choice of plaster of Paris or a brace. It is hard to say which is better. Some prefer one, some the other. Some employ both. If plaster be chosen, the knee should be fairly well, but not excessively, padded over a well fitting stockinette, and a little padding should be applied about the malleoli, and the proximal end of the thigh. The shape of the limb, that of an inverted pyramid, must be borne in mind, and the plaster must be molded carefully about the knee and calf to prevent it from sagging. At the knee the plaster should be less than $\frac{1}{4}$ of an inch thick, and elsewhere less than half that. The lighter the plaster is, consistent with strength, the less tendency it will have to sag. It is grotesque to see some plaster dressings put on to immobilize the knee, and reaching perhaps five or six inches above and below it. They should reach from the perineum to a line about one inch proximal to the tip of the lateral malleolus.

THE THOMAS BRACE.—If the surgeon elect a brace, he will probably find that the Thomas brace will serve his

purpose better than any other. It is one of the most useful braces that ever has been invented, and consists of a padded leather covered steel ring whose outline is that of a cross section of the proximal end of the thigh, and of two steel rods, running distal from the medial and lateral aspects of the ring. At their distal ends these steel rods are joined by a rubber shod sole piece. Two broad leather bands are provided to support the back of the thigh and calf, and the knee is held securely in place by a bandage, or by another broad leather band, which buckles over the front of the joint. Extension is furnished by straps fastened below to the foot piece, and running upward to buckles attached to adhesive straps on the leg.

In measuring for the ring, about an inch should be allowed for the padding. The uprights are the length of the limb from perineum to sole, plus about two inches, so that the foot will be swung clear of the ground. The sole of the other shoe must be raised an equal amount to compensate. In the later stages of the disease when traction is no longer necessary, and in the early stages as well, if we believe that fixation only is required, the uprights are fastened below to the sole of the shoe, and the high shoe on the other side is dispensed with. In any case, of course, the brace must be worn night and day.

When all active symptoms have subsided, and when the joint seems well, whatever appliance the patient has worn should not be abandoned, but should be left off at first during the night, and then by degrees during the day. If there be any return of active symptoms, or any tendency to deformity, continuous treatment should be resumed.

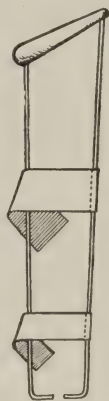


FIG. 94.—The Thomas knee splint.

Tuberculous knees that have healed in a faulty position of fixed flexion, may be straightened by linear osteotomy, or better yet by the removal of a wedge shaped piece of bone with its base forward. Osgood has devised a very ingenious operation for the correction of this deformity.¹

OPERATIVE TREATMENT

In operating on a tuberculous knee, the surgeon is guided by one of two principles; either he removes as much as possible of the tuberculous bone and synovial membrane, or he disregards completely the extent of the disease, and simply strives to produce a bony ankylosis. From reasons heretofore set forth at length, I regard the latter procedure as correct. In either case, or with whatsoever theory the surgeon operates, if he succeed in producing a bony ankylosis and in avoiding a secondary infection, he will cure the disease. The knee is an easy joint to ankylose: it is therefore an easy joint to cure.

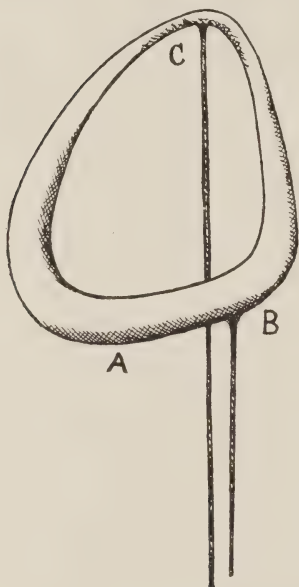


FIG. 95.—The ring of a left Thomas knee splint. C, B, the anterior portion, A, B; the perineal portion.

The approach to the knee is simple. The incision most often employed is a curved anterior, transverse one, convex distally, between the condyles, dividing the patellar tendon. Some surgeons make the incision with the convexity proximal, dividing the quadriceps tendon just proximal to the patella. Some employ a straight incision, dividing the patella transversely with a saw.

¹ OSGOOD, R. B.: "A method of osteotomy of the lower end of the femur in cases of permanent flexion of the knee joint." *Amer. J. Orth. Surg.*, 1913, xi., 336.

From this point the procedure varies with the surgeon's purpose. If he wishes to remove all the tuberculous tissue possible, he proceeds to a thorough dissection of all the synovial membrane he can reach, and to the removal of all the bone patently diseased. If his purpose is simply to ankylose, he flexes the knee acutely, dissects the soft tissues from the top of the head of the tibia, causes his assistant to retract the tissues from the head of the tibia and from the condyles of the femur, and saws off a thin slice from the head of each bone.^{2,3}

In order to make the ankylosis more secure Hibbs⁴ denudes the sides and back of the patella, and hooks it into a cavity gouged out of the head of the tibia, and the distal end of the femur. If fixed flexion was present before the operation, and if a portion at least of the lateral ligaments be spared, the joint locks fast when it is straightened.

Deep and superficial sutures close the wound.

The end of the bones is usually sawn off square. The operator plans his saw cut so that when he brings the bone ends together, the bones will be in a straight line or slightly flexed as he wishes, and bowed neither in nor out.

The difficulty of getting the two flat bone surfaces together, and keeping them in apposition, prompts some

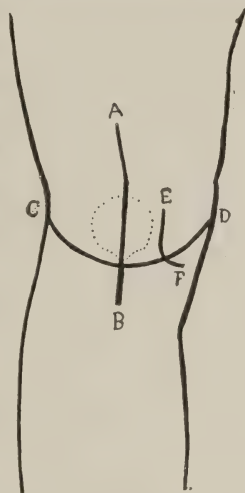


FIG. 96.—Incisions for opening the knee joint. A, B, the anterior longitudinal incision; C, D, the transverse incision; E, F, the Jones incision for removal of the torn piece of the medial meniscus.

² ELY, LEONARD W.: "Die Tuberkulose des erwachsenen Kniegelenks, etc." *Berl. klin. Woch.*, 1910, xlvii, 2062.

³ ELY, LEONARD W.: "Tuberculosis of the adult knee joint, etc." *Trans. Surg. Sect. A. M. A.*, 1910, 36.

⁴ HIBBS, R. A.: "Tuberculosis of the knee joint in the adult, etc." *N. Y. Med. J.*, 1917, cv, 922.

surgeons to practise the Fenwick operation, in which the distal end of the femur is sawn convex from before backward and the end of the tibia concave. This operation avoids one difficulty, and incurs another, and probably greater one. It leaves the bone ends in the very best possible shape for the formation of a new joint, which is just what we wish to avoid.

Some operators wire the bones together with silver, others spike or plate them. Albee recommends a bone graft. Other things being equal, metal should not be buried in the wound, on account of the danger of subsequent secondary infection. I have seen infection occur about an old spike that had been in for months, and then lost the patient eventually, after a thigh amputation.

After the wound has been sutured and dressed, the limb is encased in plaster of Paris from perineum to toes. Including the foot is wont to make the pain less. In a week or two the plaster may be removed from below the malleoli. The limb should be immobilized for about six months. Bony union does not take place for about one year.

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CHAPTER IV

TUBERCULOSIS OF THE ANKLE AND TARSUS

TUBERCULOSIS of the large joints of the extremities is peculiar in that it shows no tendency to spread from the joint where it originates, through the bone, to the joint at its other end. In other words, it remains indefinitely in and about the original joint. Tuberculosis of the joints of the ankle, tarsus and carpus does not share this peculiarity. A focus in one bone may give rise to an infection of any joint of which that bone is a component. Thereafter the disease may spread to the other bone or bones making up that joint, and then to the other bones and joints of the region. It is this peculiarity which has made tuberculosis of the carpus and tarsus so difficult to treat. Its prognosis always has been bad. On the other hand the clinical fact has been noted that tuberculosis of the ankle has not a great tendency to spread to the other tarsal joints, and that disease of the calcaneus usually remains in the bone marrow, and does not spread to a joint unless a way is opened to it by unwise surgical procedure.

THE ANKLE

Tuberculosis of the ankle is much less frequent than tuberculosis of the spine, hip or knee, and this comparative infrequency has been adduced as an argument against the traumatic origin of joint tuberculosis, for the ankle is exposed to trauma more than almost any joint in the body. The relative frequency of the disease is said to be greater in adults than in children, like disease of the smaller joints in general, but this is open to question.

As with other tuberculous joints, the proportion of persons afflicted shows a slight majority in favor of the male sex, but not enough to correspond to the greater liability to trauma.

According to Sever,¹ out of 7474 cases of bone and joint tuberculosis at the Children's Hospital, Boston, only 213 were tuberculous ankles. Of these the right ankle was involved in 108, the left in 90, and both ankles in 15.

PATHOLOGY.—The primary focus is said to be located most often in the talus, but may be in the tibia, or in the fibula. A synovial focus is assumed in some cases. The disease may spread through the talus, and involving one of the tarsal synovial membranes, may then attack the other tarsal bones, but, as has been said, this phenomenon probably is rather rare.

Abscess formation is the rule in disease in this locality, and, because of the nearness of the joint to the surface, secondary infection is almost impossible to avoid.

The condition of "fatty osteomalacia" is seen often in disease of the tarsal bones. They cut easily with the knife, and are little else but shells.

SYMPTOMATOLOGY.—Pain, swelling and limp, are early symptoms, and muscular spasm, limitation of motion, and atrophy of the leg muscles are early physical signs. The normal contour of the ankle is obliterated, the hollows behind and in front of the malleoli, and distal to them disappear, and the whole region becomes diffusely swollen. The swelling often assumes the classic spindle shape. The foot is usually held in equinus, and the patient walks upon his toes, with his knee semiflexed. Sensitiveness to pres-

¹SEVER, J. W.: "Tuberculosis of the ankle joint and tarsus." *Jour. A. M. A.*, 1910, lv, 2128.

sure is wont to be pronounced, perhaps localized more or less over the place of greatest involvement.

DIFFERENTIAL DIAGNOSIS.—This is made on the general principles already set forth, and, with the exercise of reasonable diligence and care, rarely occasions much difficulty. A sprained ankle has the traumatic history, with the symptoms following immediately, and sensitiveness distal to the lateral malleolus. Fractures have the distinct traumatic history, with positive X-ray findings.

PROGNOSIS.—This is not particularly good. Children often recover with a stiff joint after secondary infection. Resection gives a useful foot in adults, but in them secondary infection usually spells amputation.

TREATMENT.—Conservative treatment usually gives good results in children, and is not difficult to carry out. Its duration is shorter than that of disease of the hip and knee.

The joint should be immobilized in a position of adduction and right angle flexion, by a plaster of Paris dressing reaching from the bend of the knee to the toes. If sinuses be present, windows may be cut in the plaster at their openings.

If the patient walk directly upon this dressing, he will soon break the plaster to pieces, therefore we are compelled to adopt stilting in addition. This is best done with the Thomas brace, and a high shoe on the other side. Inasmuch as extension with adhesive straps is impossible in disease of the ankle, the brace is slung from the shoulders, by a looped strap, which attaches in front and behind to the thigh ring.

The Bier treatment by passive congestion, and heliotherapy are still recommended abroad particularly. They seem especially indicated in the treatment of the ankle,

tarsus and wrist. Cutting operations are rarely advisable upon children's ankles.

OPERATIVE TREATMENT.—On account of the marked tendency to abscess formation, with the risk of extension to the other tarsal joints, in disease of the ankle joint, operative measures should be undertaken in the adult as soon as a positive diagnosis is made, and here, even less than in most other localities, should one attempt the futile operation of scraping and packing. To do this is to invite disaster, for, if the disease once spread through the talus into the tarsal synovial cavities, an extensive resection, or more probably an amputation, almost always will be necessary. Our object is to produce a bony ankylosis, but this is extremely difficult in the ankle. Usually all we can secure is a fibrous one. Theoretically a bone dowel, driven up from the sole, through the talus, into the tibia, should answer the purpose well.



FIG. 97.—Whitman's incision for excision of the talus.

The operation that has given the best results is ablation of the talus, with removal of the joint surfaces of the bones with which it articulates—the Whitman operation. The incision is a curved one with the convexity distal, starting proximal to the lateral malleolus, and behind it, and running distal and forward to the anterior extremity of the talus. The peroneal tendons may be divided and later sutured. In disarticulating the talus one must keep close to the bone on the medial aspect, in order to avoid damaging the vessels passing behind the medial malleolus. If the entire foot be subluxated backward, and if the sides

of the calcaneus be freshened for apposition to the freshened malleoli, the stability of the result will be increased, and a serviceable member will be secured.

In the adult as well as in the child in secondarily infected cases, before resorting to amputation, some surgeons believe in a prolonged course of passive hyperæmia, combined with the use of Klapp's cupping, and heliotherapy.

THE TARSUS

The joints of the tarsus may be affected primarily, or secondarily by extension from the ankle joint, especially if the primary focus be situate in the talus. Secondary infection with pus producing organisms, and abscess formation are the rule, and usually occur early. According to Hahn² who has compiled statistics of 704 cases of tuberculous disease of the foot, the frequency of the disease diminishes according to the distance of the affected bone from the ankle.

The significant point in tarsal disease, besides the spongy structure of all the bones, is the extent and the ramifications of the synovial membranes. There are six or seven separate synovial cavities. The bones are bound together by ligaments running in different directions, and these again are covered by tendons, nerves and blood vessels. To ascertain the extent of the disease in the bones and synovial membranes is a physical impossibility. To attempt to eradicate it without an amputation is difficult.

The symptoms and physical signs are the same as in tuberculosis of other joints. The patient usually walks upon his heel, with his foot abducted to remove it from

² HAHN, O.: "Ueber die Tuberkulose der Knochen und Gelenke des Fusses, u. s. w." *Beit. z. klin. Chir.*, 1900, xxvi, 525.

strain. Two important points in differentiating the affection from painful flat foot are the local sensitiveness to pressure, and the signs of inflammation over the affected region. A skiagram will show roughening of the bone, with rarefaction, and irregularity of contour. Köhler's disease is also to be remembered.

TREATMENT.—In children this is almost invariably conservative. The reparative processes are vigorous, and

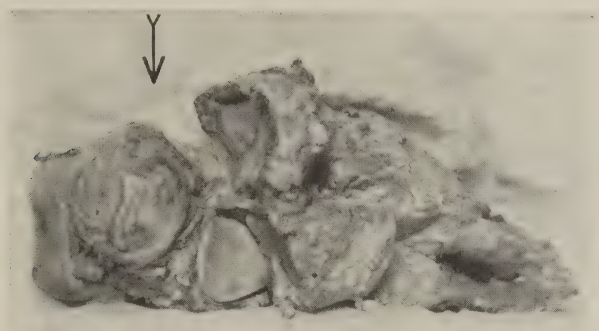


FIG. 98.—Tuberculosis of the tarsus. Talo-navicular joint laid open. Note the irregularity of the articular cartilage.

a foot with discharging sinuses, which appears to be hopelessly diseased, under conservative treatment often recovers and forms a useful member. The temptation to operate is strong, but should be stoutly resisted. In cases with secondary infection, in which drainage is poor, the foot may be immersed in a bath of normal salt solution at a temperature of 105°-110°F., for an hour or two daily. In adults, as well as in children, heliotherapy and passive hyperæmia, supplemented by the employment of Klapp's cups, may be given a faithful trial, though I am sceptical of the results in adults, and personally should not advise the treatment.

The mechanical treatment is best carried out with some such brace as the Thomas, and, if too many sinuses be not present, a plaster of Paris dressing in addition. The foot should be put up in adduction, at right angle flexion with the leg. If, on account of the sinuses, we are unable to apply plaster, we may be obliged to permit the foot to remain in a faulty attitude until the sinuses are healed. Then the faulty attitude may be gradually corrected by plaster dressings.

OPERATIVE TREATMENT.—In tuberculosis of the ankle in the adult the treatment, as in disease of other joints, is almost always operative. In a very early case, with a bone focus and no synovial involvement, an early excision might perhaps effect a cure. Later this will be difficult or impossible.

In tuberculosis of the navicular there may be involvement of two synovial cavities, one of them extensive and with a number of ramifications; in disease of the cuboid there may be involvement of three, in disease of the talus or calcaneus, of three. When two or three bones are diseased, four, five or six synovial cavities may be involved. The disease seems to run riot when once it has become diffused through the tarsus, and all temporizing measures are usually without avail.

In uncomplicated cases in the adult, wide resections have given fairly good results. Theoretically a bone graft laid down right through the diseased region should be the ideal treatment. I have never had the chance to do this operation in tuberculosis of the tarsus, but have done it in the wrist with excellent results.³

³ ELY, LEONARD W.: "An operation for tuberculosis of the wrist." *Jour. A. M. A.*, 1920, lxxv, 1707.



FIG. 99.—Tuberculosis of the calcaneus, with secondary infection.

TUBERCULOSIS OF THE CALCANEUS

This occurs fairly frequently in children, not so frequently in adults, and is peculiar in several respects. It affects, almost invariably, the anterior spongy part of the

bone, is characterized by the formation of a sequestrum, breaks down early, ruptures on the lateral aspect, and very rarely involves a joint.

The disease is a stubborn one, and its treatment is most tedious. If secondary infection have taken place, the sequestrum should be removed. The usual measures of passive hyperæmia, Klapp's cupping, heliotherapy, etc., may be tried, perhaps also plugging the cavity with an iodoform paste. Finotti has had good results with the operation of ablation of the calcaneus, and says that the ability to walk is not seriously compromised. If the tendon sheaths are badly involved an amputation will probably be necessary, according to Finotti.⁴

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⁴ FINOTTI, E.: "Ueber Tuberkulose des Calcaneus." *Deut. Zeit. f. Chir.*, 1895, xl, 450.

CHAPTER V

TUBERCULOSIS OF THE SHOULDER

TUBERCULOSIS of the shoulder is rather rare, especially so in childhood. Roughly, two clinical forms of the disease are recognized, one in which there is a production of soft granulation tissue, with formation of abscesses, and the other in which the slow tuberculous process in the marrow eats away, so to speak, the bone in the humeral head, and destroys it without abscess. The latter is more frequent, and is known as "caries sicca." The primary focus is usually located in the head of the humerus. In all my specimens this appears to have been the case.

Pain is an early symptom, felt in the shoulder, and running down the arm. Limitation of motion is present, but, on account of the mobility of the shoulder girdle on the sterno-clavicular joint, the limitation may escape the patient's notice for some time. In testing for it, the shoulder girdle should be held fast with one hand, while the humerus is moved about with the other. If the patient is simply directed to move his arm about, the vicarious motion at the sterno-clavicular joint may give a false impression to the observer. The chief limitation is in abduction, and in rotation.

Atrophy of the deltoid and of the other shoulder muscles, is an early and a very important physical sign, and is especially prominent in the dry cases. It gives to the shoulder a peculiar flattening. The roundness of the deltoid disappears, and is replaced by a more angular contour. Sensitiveness to pressure is quite marked. Fluctuation

may be detected in the moist cases. Abscesses appear at the margin of the deltoid muscle, and, rupturing, may give rise to stubborn sinuses.

Besides the other forms of shoulder arthritis of the first great type, there are two lesions of this region which deserve especial mention, namely, subacromial bursitis and acromio-clavicular arthritis. The general principles of the differentiation of the other members of this type have already been set forth.

Subacromial, subdeltoid or Duplays bursitis,^{1, 2, 3, 4,} "periarthrits" of the shoulder is, as its name implies, an inflammation of the bursa, about three centimetres in diameter, situated partly beneath the deltoid, partly beneath the acromion, and between them and the tendon of the supraspinatus muscle. It is often traumatic in origin, but often its cause can only be surmised. It is closely simulated by lesions of the subjacent supraspinatus tendon. The bursa may contain fluid, or its cavity may be obliterated by the tight fibrous adhesions which bind together its walls. Collections of lime have been described in it, but Brickner and others have shown that the lime is not in the bursa, but beneath it.

The symptomatology of subacromial bursitis is similar to that of tuberculosis of the shoulder, or identical with it. The same pain, sensitiveness, limitation of motion, and atrophy are all present. The X-ray plate may show a

¹ BRICKNER, W. M.: "Pain in the arm: subdeltoid (subacromial) bursitis." *J. A. M. A.*, 1917, lxix, 1237.

² CODMAN, E. A.: "On stiff and painful shoulders." *Boston Med. and Surg., Jour.*, 1906, cliv, 613.

³ CODMAN, E. A.: "Bursitis subacromialis or periarthrits of the shoulder Joint." *Boston Med. and Surg., Jour.*, 1908, cliv, 533.

⁴ CODMAN, E. A.: "Complete rupture of the supraspinatus tendon etc." *Boston Med. and Surg., Jour.*, 1911, clxiv, 708.

shadow corresponding to the location of the bursa, a collection of calcareous material in the same region, a slight irregularity at or near the insertion of the supraspinatus muscle, or perhaps a small tear of the greater tuberosity. Sometimes a distinct history of trauma can be obtained, such as a fall upon the shoulder, or an overstrain throwing a baseball, with the symptoms following immediately. In the absence of definite evidence a positive diagnosis between tuberculosis of the shoulder and bursitis is impossible at the first examination. If the pain and stiffness continue, sooner or later a tuberculous process, if present, will make itself known by rarefaction in the humeral head.

In acromioclavicular arthritis the pain and sensitiveness can be localized by careful examination, in the acromioclavicular joint, as distinct from the shoulder joint proper. Abduction of the arm is limited and painful, but not rotation. The X-rays make the positive differentiation. The frequency of lesions of this joint is not generally appreciated.⁵

TREATMENT.—In children immobilization may be secured by bandaging the arm to the side, and slinging the forearm from the neck. The clothing should be worn over the dressing. The author's brace constitutes a serviceable appliance for the later stages of the disease.⁶

Bony ankylosis is difficult or impossible to secure by operation, but resection usually gives good results, and is followed by tight fibrous ankylosis. The arm should be put up after the operation on an aeroplane splint, to prevent the disabling adduction that ordinarily follows opera-

⁵ SEVERS, R.: "Ueber die Bedeutung des Akromiagelenkes, u. s. w." *Archiv. f. klin. Chir.*, 1914, cv, 418.

⁶ ELY, LEONARD W.: "A new brace for the shoulder joint." *Med. News*, 1904, lxxxv, 160.

tions on the shoulder. If ankylosis in abduction can be secured, the rotation of the scapula will permit excellent function in the joint. For the same reason immobilization in internal rotation should be avoided.

Several operations for resection have been devised. The simplest and the best for routine work is perhaps that through the anterior incision with the arm in abduction on a board. The incision, about twelve centimetres long, runs distal from the clavicle along the anterior border of the deltoid. The operator identifies the median cephalic vein, and retracts it or ties it between ligatures, carries his dissection between the deltoid and the pectoralis major, anterior to the biceps tendons, to the joint capsule, and slits the capsule widely at right angles with the line of the joint. He then dissects subperiosteally all the tissues from the head and from the tuberosities, the assistant rotating the arm meanwhile first in one direction, and then in the other. The assistant dislocates the head through the wound, and the operator saws it off. If he believe in the efficacy of the removal of tuberculous tissue, he removes all he can reach with knife, scissors, gouge and chisel; otherwise he contents himself with the removal of the head and the articular surface of the glenoid. The operator finally closes the wound with deep and superficial sutures, dresses it, and puts the limb on the splint so applied that, when the patient stands, his hand will be about on a level with his mouth. The splint should be worn for two or three months.

Kocher's posterior incision offers no particular advantages in tuberculosis of the shoulder, and is much more difficult.

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CHAPTER VI

TUBERCULOSIS OF THE ELBOW

THE disease is said to begin most often in the ulna, less frequently in the humerus, and least frequently in the head of the radius. Primary synovial disease seems to be fairly common. In one of my specimens the only bone lesion discovered was a bunch of tuberculous granulations sprouting up through the articular cartilage of the radius. Abscess formation is frequent, and bursting of the abscess can with difficulty be prevented.

The *pain* is felt in the elbow, and perhaps shoots down the forearm. *Swelling* is an early symptom, and is usually diffuse. If fluid be present, it is manifest posteriorly at the sides of the triceps tendon. The muscles of the arm and of the forearm shrink, and this atrophy, coupled with the swelling of the joint, causes the classic spindle shape of the elbow, so often seen in the elbow, knee and ankle. On the other hand, swelling may be absent entirely in the slow, dry, fibrous cases.

The forearm is semiflexed, and midway between pronation and supination. All motions in the elbow may be limited, or the limitation may be confined to flexion and extension, rotation remaining free. In the latter case, the radio-humeral pouch is assumed to be intact.

The differential diagnosis rarely causes much difficulty. Leaving out of consideration the general diagnostic points already detailed, for some unknown reason a slow, chronic, uniarticular lesion in the elbow joint is usually tuberculous.

TREATMENT.—Here as in other joints, the treatment is conservative in children, radical in adults.

CONSERVATIVE TREATMENT.—Some surgeons put their trust in heliotherapy and passive hyperæmia, slinging the forearm from the neck during the course of the treatment. Most surgeons rely upon immobilization in plaster of Paris. The elbow is put up in plaster, in flexion at a right angle or slightly beyond, and is kept in plaster until it is well. This position, enabling the patient to get his hand to his face, is the most serviceable for the general run of patients, but the rule may be modified in certain circumstances. Adults should choose the attitude in which a stiff elbow would be most useful to them in their business.

In order to get the joint into the required position, ether or gas may be administered, or, better yet, the method of Thomas may be adopted. Thomas slung the wrist to the patient's neck, pulling the sling tight enough to make him bend his neck down toward his hand. As this position is uncomfortable, the patient gradually straightens up his head, flexing his elbow to that extent. This procedure is repeated daily until the required flexion has been secured.

If sinuses be present, windows may be cut in the plaster. Carrying the plaster bandage repeatedly up and down the extensor surface of the arm and forearm, avoids the piling up of the plaster in the reentrant angle, which occurs with the ordinary method of application. The dressing reaches from the axilla to the wrist, and is reinforced by an ordinary sling about the wrist.

OPERATIVE TREATMENT.—The operative treatment of a tuberculous elbow is not, as a rule, very satisfactory, and this is probably because the joint is a difficult one to ankylose. A bony ankylosis in an attitude of right angle flexion, would give a serviceable member. The customary treatment is resection. This gives a rather loose joint, which appears for a time to be well. Then, with re-forma-

tion of the synovial membrane, the disease lights up afresh, and compels another resection, with the sacrifice of more bone. The resection is repeated, perhaps several times, until at length, with the ends of the bones sclerosed and tied loosely together with fibrous tissue, cure results. A flail joint is not very useful. If one expects nature to build up an elbow joint anew after a resection, as in Ollier's classic and oftquoted case, one will be disappointed. (See the section on bone formation in the first chapter.)

The resection is done subperiosteally, and the operator must beware to keep close to the bone throughout the operation, anteriorly especially to avoid the vessels and nerves, medially especially to avoid the ulnar nerve as it passes behind the medial condyle. Some surgeons employ two posterior incisions, some Kocher's incision, and some the Z-shaped incision of Ollier, but most prefer the single, long posterior incision.

The single posterior incision, about twelve to fifteen centimetres long, is made over the middle of the olecranon, and is carried down to the humerus and ulna, opening the joint. The olecranon, the posterior surface of the ulna for a short distance distal to it, and the posterior surface of the distal end of the humerus are skeletonized. The olecranon process is removed with a few strokes of the mallet and chisel, giving access to the joint. After the condyles of the humerus and the end of the ulna have been denuded of periosteum, they are removed with saw or chisel. The head of the radius can be reached through the opening in the lateral wall of the wound, and it also should be removed. The wound is closed with deep and superficial sutures. Some authorities have maintained that if the joint be put up in full extension for a week or so, and if the position

then be changed to right angle flexion, bony ankylosis will ensue. The custom is to put it up in flexion.

There is a good opportunity, in disease of the elbow, for the employment of ingenuity to ankylose the joint with a bone dowel or inlay graft. At the suggestion of one of my students I attempted to drive a dowel through the olecranon into the medial condyle, but evidently missed the condyle, for motion was present when the plaster was removed six weeks later. It might be possible to run a strut from the olecranon to a niche just proximal to the olecranon fossa of the humerus. Of course, if the radio-humeral joint is intact, all ankylosing work should be done between the ulna and the humerus, so as not to compromise the important motion of rotation. To lay down a graft between the head of the radius and the lateral condyle should be a comparatively simple matter.

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CHAPTER VII

TUBERCULOSIS OF THE WRIST

TUBERCULOSIS of the wrist is rare in childhood, somewhat less so in the adult. The primary focus may be in any one of the bones entering into the formation of the joint, or possibly in the synovial membrane. The most frequent starting place is said to be the head of the radius. Owing to the great extent of the synovial membrane, and to its many ramifications, the disease, once started, tends to spread widely, and to involve the synovial cavities one after another in a manner described in the section on tuberculosis of the tarsus. Abscesses form early and break down, leaving infected sinuses leading down to the joint. The tuberculous inflammation is especially likely to spread to the tendon sheaths passing over the joint, and the resulting tuberculous synovitis adds severity to the disease.

The wrist is often swollen, and usually it is in slight flexion and in pronation. Sensitiveness is present over the site of the disease, and sometimes fluctuation and muscular atrophy. A boggy swelling is more frequent than a fluctuating one. Motion of the joint is restricted in every direction, and, as a rule, quite painful. If the tendon sheaths are involved, the motions of the fingers also are restricted.

In the matter of differential diagnosis only two things need particular notice, namely tuberculosis of the tendon sheaths, and fracture of one of the carpal bones, most often the navicular.

In tuberculous tendovaginitis, the swelling is more superficial, in a manner of speaking, and is localized to the region of the tendon sheaths. It moves up and down with the movement of the fingers. The Röntgen rays



FIG. 100.—Tuberculosis of the wrist, treated with plaster of Paris dressings for several years. Author's operation in August 1919. Positive guinea pig test with some of the material removed from the joint.

reveal no disease in the bones of the wrist, and motion at the wrist is unrestricted. Essential tuberculosis of the tendon sheaths is rare.

In fracture of the carpal bones, an injury immediately antedates the trouble, and the sensitiveness and swelling are limited to a particular region. The Röntgen rays show the fracture.

TREATMENT

For conservative treatment, the usual expedients are employed—heliotherapy, passive hyperæmia, immobilization, etc. Iodoform injections have their advocates here as in other joints. If immobilization be chosen, as I think it should be invariably in children, the hand should be put



FIG. 101.—The wrist shown in Fig. 100, eleven months after operation. Apparent cure with a stiff wrist.

up in a position of slight superextension. This attitude gives the best function. The plaster reaches from the bend of the elbow to the metacarpophalangeal joints, and is cut out well at the base of the thumb. Free motion in the fingers and thumb is to be encouraged. The prospects of a cure by conservative measures are not particularly bright.

Operative treatment heretofore has also been disappointing in disease of the wrist. One resection follows

another, until at last the joint becomes secondarily infected, and an amputation is necessary to save the patient's life. The only good results seemed to follow wide resection, with removal of all the carpal bones, the end of the radius, and the bases of the metacarpals. I have employed the following operation in two cases with beautiful results, and recommend it strongly. It has a strong theoretical foundation, and the results indicate that the theory is correct.¹

The longitudinal, median, posterior incision, about fifteen centimetres long, runs from a point about five centimetres proximal to the end of the radius to one about five centimetres distal to the proximal end of the third metacarpal. The incision is deepened, and the extensor tendons are retracted.

The periosteum over the distal end of the

radius, and the proximal end of the metacarpal is incised longitudinally for about two centimetres, and pushed aside, baring the bones. With a motor saw a gully about five millimetres wide is cut in the wrist, running into the third metacarpal and the radius for the distance of one

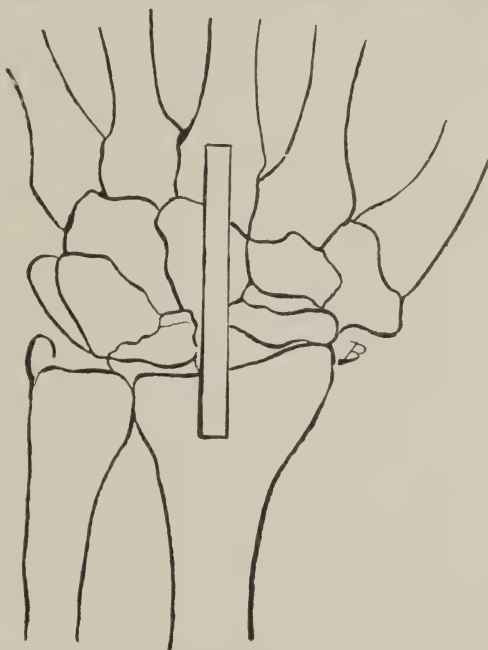


FIG. 102.—Author's operation with bone graft for tuberculosis of the wrist.

¹ELY, LEONARD W.: "An operation for tuberculosis of the wrist." *Jour. A. M. A.*, 1920, lxxv, 1707.

centimetre. The gully is cut quite superficial in the carpal bones but deep in the radius and metacarpal. A graft about three centimetres long and five millimetres wide, with periosteum attached, is then removed from the antero-medial cortex of the tibia, and is fitted into the gully. When the hand is forced into superextension the graft locks fast. Deep and superficial sutures close the wound. The hand is put up in plaster of Paris, while care is taken to maintain the superextension. After the plaster has partially set, it is slit up longitudinally together with every turn of the bandage underneath, for the postoperative swelling is considerable. The fingers and thumb are left free, and early motion of them is enjoined. The plaster stays on for three or four months, so as to give time for the formation of a firm bony bridge between the radius and the metacarpal.

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CHAPTER VIII

TUBERCULOSIS OF THE SACRO-ILIAC JOINT

TUBERCULOSIS of this joint is very rare. Its occurrence in childhood is unknown. The male sex is said to claim a small majority of the patients, and among women the affection is especially apt to occur during pregnancy.

The synovial membrane is comparatively small in extent, but the great masses of cancellous bone on either side of the joint afford the disease a good field for extension. The ligaments above and below are strong and thick, but those in front and back are thinner, and through these the abscess, whose formation is the rule in disease of this joint, may break anteriorly into the pelvis, or posteriorly, or both anteriorly and posteriorly. Of fifty-nine abscesses tabulated by Van Hook¹ twenty-one, or 38.2 per cent. were extrapelvic, and thirty-eight, or 61 per cent. were intrapelvic. Secondary infection of the abscess almost always takes place, and, when it occurs, the disease has a free field in the sacrum and ilium, giving rise to many burrowing sinuses and to marked constitutional involvement.

Pain is the first symptom of which the patient complains, felt either at the seat of the disease, or in the buttock, or shooting down the lower extremity. It is wont to manifest itself as a severe sciatica, and to be increased by motion, that is, by walking, coughing or sneezing. It can be brought out by pressing the wings of the ilia together, or by the Kernig manipulation.

On account of the pain the patient stands with his

¹ VAN HOOK, W.: "Tuberculosis of the sacro-iliac joint." *Annals of Surg.*, 1888, viii, 407; 1889, ix, 35 and 115.

weight thrown over on the sound leg, giving a pronounced lateral curvature to the spine. The Kernig sign is positive. *Swelling*, on account of the nearness of the joint to the surface, appears early. Local sensitiveness to pressure can be detected posteriorly, or anteriorly in the vagina or rectum. The fluctuation of an abscess can be felt in either one of these places or in both.

The prognosis has been distinctly bad. Owing to the superficial situation of the disease the abscess is extremely liable to early infection, and infection here usually means death. There is almost no limit to the extension of the disease through the pelvic bones, and the sinuses burrow in every direction.

The treatment always has been unsatisfactory. Text-books pass over it rather cursorily, and necessarily so. One sees so few cases, even in a large clinical experience, that one hardly has a chance to formulate any well defined method.

Not much is to be expected from conservative measures. Whitman recommends a plaster spica, or a double Thomas hip splint combined with crutches. A high shoe should be worn on the well side so as to relieve the affected joint from weight bearing.

OPERATIVE TREATMENT.—Until recently the outlook after operation on a tuberculous sacro-iliac joint was decidedly gloomy, and in probably no joint was the error of the old theory of operating more clearly demonstrated. The surgeon, attempting to remove all the diseased tissue, of course failed. The wound broke down, became infected, and death usually was not long delayed. Recent results, based solely on the theory of building a bony bridge across between the ilium and the sacrum, have been more successful.

Hibbs² makes a curved incision along the posterior third of the iliac crest, continued down over the sacro-iliac joint, and reflects the thick flap of skin and subcutaneous tissues. He then chisels a large bone flap from each bone,

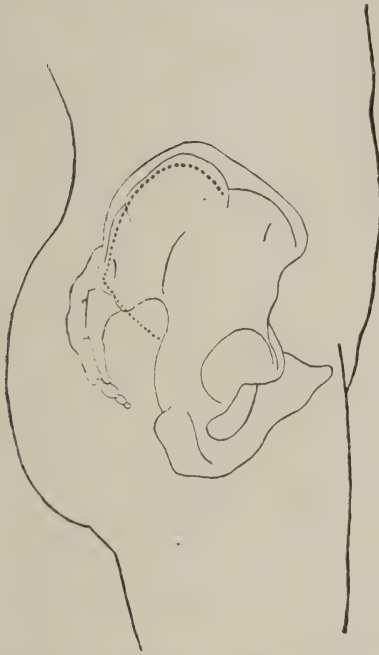


FIG. 103.—The Smith-Peterson approach. The dotted line represents the incision in its relation to the ilium; the curved limb of the incision extends from the posterior superior spine two-thirds of the distance to the anterior superior spine. The straight limb runs from the posterior superior spine in the direction of the fibres of the gluteus maximus muscles for a distance of approximately three to four inches.

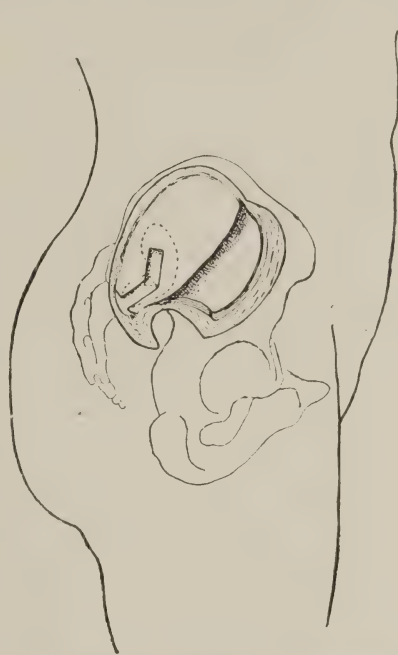


FIG. 104.—Flap reflected by subperiosteal dissection from lateral surface of the ilium. The dotted line shows the sacro-iliac joint projected on the lateral surface of the ilium with windows cut in two different planes.

and turns it over on the denuded surface of the other. The iliac flap includes the external lamella of the posterior part of the ilium with most of the posterior superior spine and the attached periosteum and soft tissues. The

²This description is from a personal communication of Doctor Hibbs. The operation has not been published as yet.

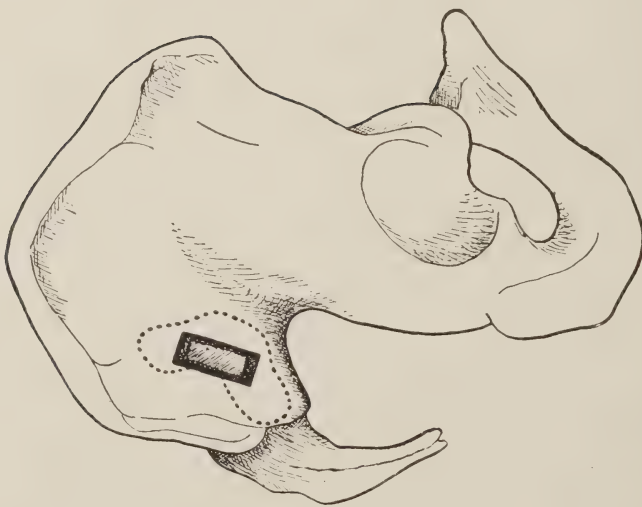


FIG. 105.—The dotted line represents the sacro-iliac joint projected on the lateral surface of the ilium. Window removed as described for cases of tuberculosis or of relaxation of the sacro-iliac joint. Note that the window is well posterior to the median gluteal line and just above the sacro-sciatic notch.

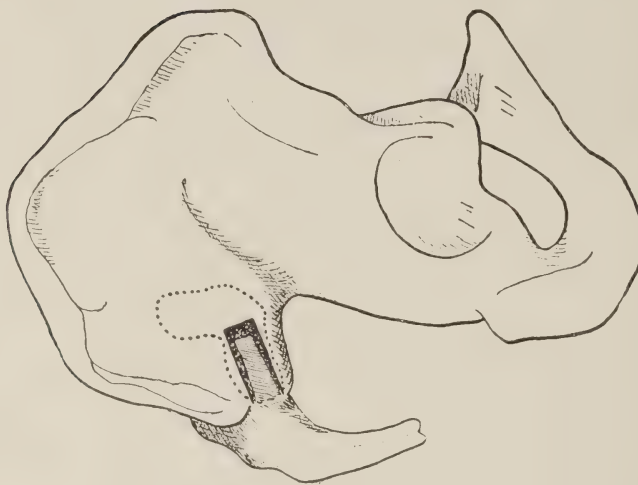


FIG. 106.—The dotted line represents the sacro-iliac joint projected on the lateral surface of the ilium. Window removed as described for cases of osteomyelitis: from the posterior border of the sacro-iliac joint between the posterior superior and the posterior inferior spines. It runs anteriorly parallel with the sacro-sciatic notch.

sacral flap includes the external lamella of the proximal, lateral part of the sacrum, with its periosteum and attached soft tissues.

Albee employs a bone graft between the sacrum and ilium.

Smith-Peterson has recently devised an extremely ingenious method of approach to the joint. His incision has two arms, one run-



FIG. 107.—Window removed from ilium down to the sacro-iliac joint.



FIG. 108.—Cortex removed from portion of the sacrum opening the cancellous bone.

ning from the posterior superior spine of the ilium, along the crest for about fifteen centimetres. It is carried down to the bone. The other arm runs from the posterior superior spine distally and anteriorly in the direction of the posterior inferior spine toward the sacro-sciatic foramen for about fifteen centimetres. This arm is

carried down to the bone, splitting the fibres of the gluteus maximus, in whose direction it runs. The thick flap of skin, fascia, periosteum and muscle is then reflected distally and anteriorly, the fibres of the gluteus maximus and medius being separated from the bone close to their origin from the ilium. This procedure lays bare the whole posterior portion of the external surface of the ilium.



FIG. 109.—The block of bone from the ilium countersunk into the sacrum.

The operator then removes from the ilium at a place corresponding to the location of the distal part of the

sacro-iliac joint, a bony block about 1 x 3 centimetres in diameter right down to the sacro-iliac joint. The ilium here is often very thick. Next he goes through the sacro-iliac joint, and removes a corresponding piece of bone from the sacrum. Now he countersinks his block of iliac bone into the hole, so that it passes through the joint into the sacrum. The wound is closed, and a double plaster of Paris spica is applied for two months, then a single spica.

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CHAPTER IX

TUBERCULOSIS OF THE FINGERS AND TOES

TUBERCULOSIS of the bones and joints of the fingers and toes—tuberculous dactylitis—often possesses some features that distinguish it from the disease as ordinarily observed elsewhere in the skeleton. In adults it may occur as an ordinary arthritis, like a tuberculous arthritis anywhere. In children it is seen as a tuberculous myelitis of the shafts of the metacarpals, metatarsals or phalanges. This is the so-called “spina ventosa.”

TUBERCULOUS ARTHRITIS is rather a rare affection. I do not remember to have seen more than two or three cases in many years of clinical work, and have but two laboratory specimens. One of them, an amputated finger of an adult, shows far advanced synovial disease, with secondary infection, and extension of the process to the bone marrow. Apparently it was a primary synovial case. The synovial membrane hangs in long streamers like moss from a tree. The patient died from pulmonary tuberculosis shortly after the operation.

Another specimen represents tuberculosis of the metatarso-phalangeal articulation of the great toe, also in an adult. Its origin is impossible to determine, though the bone is badly involved. The treatment in this case was excision and it was followed by a cure.

The structure of the bones entering into the articulations of the fingers and toes of an adult is that of the long bones generally, namely, cancellous bone at their ends, and dense bone with a medullary canal in the shafts. The



FIG. 110.—Tuberculosis of the metatarso-phalangeal joint.

disease has no tendency to spread from one joint through the fatty marrow of the shaft to another. This fact is another link in the chain of evidence in behalf of the theory of the relation of the lymphoid marrow to tuberculosis.

The symptoms are pain, swelling, sensitiveness, limitation of motion, etc. The diagnosis as a rule presents no peculiar difficulties, and is made on the points hitherto detailed. When the disease occurs in the metatarsophalangeal joint of the great toe (I have never heard of a case in the other toes), it might be mistaken for gout. In gout there are almost always other evidences of the same disease elsewhere in the body, chalky deposits are present about the joint, and the Röntgen rays show the punched out areas in the bone near the joint.

TREATMENT.—Conservative measures are hardly to be advised. A finger is of little importance. In the great toe the joint may be excised at the earliest possible moment, with the hope of saving a member that is of considerable importance in locomotion. The same treatment may be applied to a finger, but a stiff, deformed finger is not of much service, and most of us personally would probably prefer an amputation.

SPINA VENTOSA.—Tuberculosis of the marrow of the shaft of the bones of the fingers, much more rarely of the toes, is fairly frequent in childhood, and in its essentials appears to be the same as tuberculosis of the shafts of the long bones of the extremities, a great rarity in this country, but evidently quite common abroad. The disease has practically no tendency to attack the joints. Here we see a startling evidence of the truth of the statement that all definite infectious processes in bone, are alike in their fundamentals, and only differ in their details. We are reminded of suppurative osteomyelitis, which, when it involves the joints, has little tendency to spread to the shafts, and *vice versa*.

The tuberculous process in the marrow of the shaft, is prone to break down early, become secondarily infected,

and establish a communication with the outside, resulting in the formation of a sinus which is difficult to heal. Some authorities affirm that the disease sometimes heals without breaking down, and without sinus formation,¹ but I do not remember ever to have seen such a case.

New bone is said to be laid down in the periosteum, and through the hole in the cortex a probe can be passed into the diseased, apparently enlarged marrow canal. Some writers, basing their opinion upon radiographic evidence, deny the existence of new periosteal bone. Whether or not they are correct, the phalanx takes on a peculiar, enlarged, flask-like distended appearance (whence the name).

Several phalanges may be affected, though not in the same finger, and this multiplicity of the lesions distinguishes the disease from tuberculosis in bone as ordinarily observed, and has given rise in some of the cases to a strong suspicion of syphilis, a suspicion strengthened by the bone production in the periosteum.

DIFFERENTIAL DIAGNOSIS.—To distinguish tuberculous from syphilitic dactylitis is often an extremely difficult matter. The differentiation is to be made on the general principles heretofore set forth.

TREATMENT.—Conservative treatment will usually be found preferable to radical. Sterile dressings, cleanliness, heliotherapy, passive hyperemia with Klapp's suction cups, will be found useful. Some authorities recommend radical measures. The diseased bone may be dissected out subperiosteally and primary union of the wound can be secured, but in my experience little is gained in this way. As soon as the bone re-forms, the wound breaks down, and the trouble starts up as bad as ever. Some surgeons have

¹ TILLMANN'S: "Lehrbuch der allgemeinen Chirurgie." Leipzig. Robert Veit u. Co., 1892.

reported success with bone transplanted from the tibia, or from a toe.

Tuberculosis of the ribs is of fairly frequent occurrence. It is manifest ordinarily by a comparatively painless swelling, usually fluctuating, near the junction of the rib with its cartilage. It may be treated by aspiration and injection, heliotherapy, etc., but usually resection of a portion of the rib will be necessary.

Tuberculosis of the sternoclavicular articulation, of the pelvic bones, of the scapula, etc., are more or less curiosities. They are diagnosed and treated on the principles already laid down. The same may be said of tuberculosis of the bones of the head and face, except that the disease is fairly frequent in some of them, such, for instance, as the mastoid process of the temporal.

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SECTION VI

OTHER FORMS OF ARTHRITIS OF THE
FIRST GREAT TYPE OR GROUP

CHAPTER I

COCCIDIOIDAL GRANULOMA

TO POSSESS a knowledge of the pathology and symptomatology of tuberculosis is to possess a knowledge of the pathology and symptomatology of all the members of the group of which it is so conspicuous an example. Their fundamental characteristics are the same, and, while each possesses individual peculiarities which identify it with reasonable certainty in the great majority of cases, in no instance is the identification complete without the demonstration of the causal organism. In some of them, as, for instance, tuberculosis and coccidioidal granuloma, this demonstration can be made, in others it is often impossible, and the cases recover, or progress for years, under a diagnosis that must never be regarded as anything more than presumptive. The ætiology of some of them is known; the ætiology of others has almost been established; the ætiology of others is absolutely unknown. To take refuge in such terms as "faulty metabolism," "diatheses," or "rheumatoid" will not help us.

In dealing with cases in this great group, one interesting fact is always to be borne in mind: They all look alike, and the clinical diagnosis is never made from an examination of the joint itself, but from the history and from an examination of the entire patient.

The members of the group which chiefly interest us are:

1. Syphilitic and tuberculous arthritis. These have already been considered. The diagnosis of tuberculosis can be made positively. That of syphilis is usually presumptive.

2. Coccidioidal Granuloma. In this a positive diagnosis is possible.

3. Chronic arthritis due to the diplostreptococcus. The causal organism has been demonstrated in some of the suspected cases, in others it has not. With improved technique, the demonstrations have become more frequent. The domicile of the organism is presumed to be, in the great majority of cases, the tonsil or the deep urethra, but positive proof is lacking. A chronic osteomyelitis at the roots of the teeth is also thought by most observers to be a possible primary focus, but I believe that this lesion causes an entirely different type of disease, and is never the cause of this type of chronic arthritis.¹

4. Chronic, progressive, multiple arthritis, which differs from the preceding in its steady and remorseless progression, its production of fibrous ankylosis in almost every joint in the body, its crippling flexion deformities, and in the fact that its cause is absolutely unknown.

5. Still's Disease, the multiarticular form as it occurs in children.

COCCIDIOIDAL GRANULOMA

The *öidium coccidioides* is a yeast fungus, in this country almost exclusively domiciled in the San Joaquin valley in California. Practically all the patients afflicted with the disease which it causes, have lived at some time in or about the town of Los Baños. It produces in the bone marrow an inflammation whose symptomatology and clinical course are identical, up to a certain point, with those of tuberculosis. The effect of this inflammation upon the bone and upon the joint tissues is also the same as is that

¹ ELY, LEONARD W.: "The great second type of chronic arthritis." *Archives of Surg.*, 1920, i, 158.

of a tuberculous inflammation, so that the Röntgen picture of the one cannot be distinguished from that of the other. The likeness of the two diseases is so great that even the California surgeon usually mistakes the rarer affection for the more frequent, and only becomes aware of his mistake when the bacteriologist informs him of the presence in a cold abscess of the characteristic spores.

"The parasitic organisms which cause the disease are easily found in the tissues, in the pus and in the sputum. They appear as spherical bodies about 30 microns in diameter with double-contoured capsules and a slightly granular protoplasm which is sometimes vacuolated. The capsule is highly refractile, and in some cases seems to have short knobs or prickles on the outer surface. A true nucleus has never been demonstrated. In the tissues these spherical bodies are found in the diseased areas in large numbers, usually within the tubercles, and frequently inside the multinuclear giant cells. In the walls of the abscess cavities and in the pus they are usually very numerous, and can be demonstrated with little difficulty. In the pus or in the sputum they are best seen in a fresh specimen prepared by placing a cover-slip over a drop of the material on a glass slide. If examination is made with but little light, the spherules stand out prominently and are easily distinguished from cells and from the myelin droplets which are also usually present. The bodies stain poorly with the ordinary staining methods and are very apt to be overlooked in the stained smear.

"Reproduction within the tissues occurs by a process of endosporulation, and the organism differs from the blastomyces in that true budding has never been observed. The spores develop by a division of the protoplasm within the capsule until a large number, a hundred or more, are

formed, and when mature, they are so closely packed together that they assume various shapes. They are released through a rupture in the capsule of the parent body, and it is not at all uncommon to find a number of the empty envelopes in a specimen of pus. The development of the adult forms from the spores has not been definitely traced, because the spores are so small and they stain so poorly that it is impossible to follow the changes that occur after they escape from the parent body. However, Ophüls was able to find a few cases in which spores seemed to mature within the body of the parent before rupture of the capsule liberated them, and he believes that the adult forms develop directly from the spores by simple growth, without further change than the formation of the relatively thick, double-contoured capsule."²

There are a few slight differences in the clinical behavior of coccidioidal granuloma and tuberculosis which, if the surgeon be on his guard, may possibly help him to distinguish the two. The former is perhaps not quite so prone to be confined to the ends of the long bones, nor to correspond to the distribution of the lymphoid marrow. The lesions are more likely to be multiple, and involvement of the viscera is frequent. The pain, muscular spasm, and deformity are perhaps not so great, in proportion to the damage to the bone as revealed by the X-rays.

Treatment is usually of no avail. All the reported cases, except one, terminated fatally, and this is said to have recovered under the administration of iodide of potassium. The cold abscesses should be aspirated under strict asepsis, and every effort should be made to prevent the establishment of a communication between the focus of the disease and the outside air.

² DICKSON, ERNEST C.: "Oidiomycosis in California, with especial reference to coccidioidal granuloma." *Arch. Inter. Med.*, 1915, xvi, 1028.

CHAPTER II

CHRONIC DIPLOSTREPTOCOCCIC ARTHRITIS

ÆTIOLOGY.—Upon considerable evidence, clinical and experimental, the diplostreptococcus domiciled in the tonsil, deep urethra, and occasionally also in other organs, is assumed to be the cause of a form of chronic arthritis, whose clinical manifestations vary, but whose pathology and symptomatology are fairly well defined. This is the “infectious” arthritis of Goldthwait, and part of the “proliferative form” of Nichols and Richardson, and of the “rheumatoid” arthritis of the English.

Sometimes the fluid and joint tissues are sterile, sometimes they yield a diplococcus, sometimes a streptococcus “rheumaticus,” *sive* “viridans,” *sive* “hæmolyticus.” Pure cultures of these organisms often produce in laboratory animals a similar form of arthritis, and from the affected joints of these animals the organisms can be recovered. Rosenow is authority for the interesting statement that all these organisms are really one and the same, simply changing their form according to the conditions under which they find themselves. They can be recovered from the mouth of normal individuals, and in ordinary circumstances are harmless, but when growing under pressure, as, for instance in the deep crypts of the tonsil, or in the seminal vesicles they may take on pathogenic properties. When growing in the tonsil, their virulence is affected by changes in the secretion of the mouth; hence the well recognized influence of the emotions and of digestion in the ætiology of the disease. Chilling of the surface of the body, perhaps by lowering the resistance, seems to be a predisposing factor.

When the focus is in the deep urethra, this form is often called chronic gonorrheal, or chronic gonococcic arthritis, but it is doubtful if the gonococcus, even if present, is ever responsible for these old, chronic arthritides. The cause is probably the other organism, grafted upon the original infection. In other words we have here again a case of secondary infection. The gonococcus causes an acute arthritis, and its presence in the joint is fleeting. It does its work quickly, and disappears.

We find, therefore, in patients with this type of arthritis evidences of chronic tonsillitis, of chronic prostatitis, of seminal vesiculitis, etc. The suspicious tonsil is as a rule not the large, succulent, hypertrophied one as might be thought, but the small, fibrous, buried one. Sometimes the patient will give a history of frequent attacks of sore throat; perhaps more often he will give a history of having had attacks many years previously, even of attacks of quinsy, but without further throat trouble in years.

Chronic infection of other organs, such as the gall bladder and the female genitals, has also been held responsible for this form of arthritis. This may be true, but I am somewhat sceptical of it.

PATHOLOGY.—We have not the same abundance of pathological material of cases of this disease as we have of tuberculosis, and our knowledge of the pathology therefore is not so nearly complete, but from what we do possess it appears that the essentials of the pathology of the two diseases are much the same. There is the same proliferative inflammation in the marrow, and in the synovial membrane, with a similar effect upon the bone and cartilage, but, apparently, in the majority of cases, in contradistinction to tuberculosis, the burden of the attack is borne by the synovial membrane rather than by the marrow. The

bone in the vicinity of the joint is absorbed to a greater or less degree, and probably the cartilage is attacked from beneath by the granulation tissue in the marrow, and perforated. Adhesions form between the marrow and the synovial membrane, and between the marrow of one bone and the marrow of the other.

A hyperplastic inflammation may take place in the synovial membrane, with an abundant villous formation. This is the so-called villous arthritis of some writers. In such a case a fluid is poured out into the joint, usually serous in its nature. On the other hand this reduplication of the synovial membrane and villous formation, may be absent, as in tuberculosis, and fibrous changes may predominate. In such case the joint will be dry, and shrunken rather than swollen. Naturally in the latter case, the adhesions in the joint would be much more in evidence than in the former.

The disease shows a marked predilection for certain joints. The spine may be affected in whole or in part. A localized involvement, as occurs in tuberculosis, is, at best, very rare. The typical lesion is an arthritis of all the spinal joints except the two uppermost. The hips and shoulders usually escape, the wrists, knees, ankles, and tarsus are frequently involved. A multiple arthritis of the finger joints is characteristic of some cases, especially of those caused by disease in the tonsil. Strangely enough, and in contradistinction to the second great type of arthritis, the metacarpophalangeal, and the proximal interphalangeal joints are attacked, while the distal interphalangeal joints escape. Temporo-mandibular arthritis is fairly frequent.

Secondary infection and suppuration are extremely

rare, if they ever occur. I do not remember ever to have seen abscess formation in one of these cases.

The duration of the disease varies. It may be fleeting, it may persist indefinitely, or it may clear up at any time, following treatment. It may disappear and recur, especially if its cause have not been removed. Complete recovery may follow an attack, or the fibrous adhesions may cause an ankylosis more or less complete, according to their amount and density. This fibrous ankylosis may be permanent, or with time it may slowly yield and allow a fair degree of motion, especially if the original cause have been removed. In the last analysis the degree of permanent ankylosis seems to depend upon the amount of damage to the joint cartilage.

COURSE.—One joint alone may be affected, but if the disease last any length of time, practically invariably it shows a multiarticular tendency. This may be said to be characteristic of it. Sometimes only two or three joints are involved, at intervals, or all together. If many joints are involved, more or less of a tendency to symmetry is wont to be present, especially in the case of the hands and feet. The inflammation does not disappear from one joint as the next one is attacked, as it does in acute inflammatory rheumatism, but persists. The progression may be more or less steady, or it may be characterized by remissions.

SYMPTOMATOLOGY.—The symptoms and physical signs are those common to all the members of this type of arthritis, namely, pain, usually swelling, sensitiveness to pressure, disability, limitation of motion, and muscular atrophy.

The pain varies. It may amount to no more than a feeling of slight discomfort and stiffness, or it may be very severe. Roughly, one might say that it about equals the pain of an ordinary case of synovial tuberculosis, but rarely

equals that of joint tuberculosis with bone involvement. The pain is rather severe in the acute exacerbations, but not so great during the remissions of the disease. It is worse, of course, on motion.

If swelling be present, it is as a rule due to fluid in the joint rather than to thickening of the synovial membrane, though especially in the fingers, the soft parts may be decidedly thickened. The synovial membrane is almost invariably sensitive to pressure. When the disease is in the lower extremity, the patient limps. The limitation of motion ranges from a slight limitation at extremes, to an almost complete absence of motion. It is usually marked, and is due to the fluid in the joint, to the thickened synovial membrane, to the adhesions, both in the joint, and in the neighboring tendon sheaths, and to the muscular spasm. Muscular spasm, and muscular atrophy may be prominent, but not as prominent as in tuberculosis.

DIAGNOSIS.—The pain, swelling and limitation of motion make the diagnosis of an arthritis a simple matter. The next step is to ascertain in which type of arthritis the case belongs. This is done on the symptoms, and chiefly on the X-ray evidence. An arthritis of the second great type shows a lower grade of inflammation, as a rule less pain and a range of motion that is practically painless, and is only limited at extremes, and then only by the mechanical obstruction of the new bone formation. The X-ray plate shows the new bone formation at the line of insertion of the capsule, with spurring and lipping, in the second type.

Having ascertained the type, we next try to establish the identity of the particular member of the type. The other members which most merit consideration, are tuber-

culosis, syphilis, and the hopeless progressive form later to be described.

Tuberculosis is to be suspected in a slow, chronic, painful inflammation in a single joint, more or less steadily progressing, and with no appreciable tendency to recovery without treatment. The muscular spasm and the muscular atrophy, probably on account of the more extensive disease in the bone, are greater than in the form of arthritis under consideration. The history may help us, and the physical examination of the patient himself, but we must not permit ourselves to be too much swayed by their result. A patient with pulmonary tuberculosis may have a diplostreptococcus arthritis, and a patient who gives a history of many Neisserian infections and frequent sore throats, may have a tuberculous joint. The only sure test is by the demonstration of the causal organism. This may be impossible in a diplostreptococcic inflammation, but it is almost invariably possible in a tuberculous joint.

Syphilitic joints rarely show the same amount of fibrous adhesions as this form, but personally I know no way to differentiate the two except by the therapeutic test. The history, the glandular enlargement, the Wassermann and Noguchi tests are all suggestive but not conclusive. Of course, if the patient give a distinct history of syphilis, and show definite stigmata of the disease, the presumption of a syphilitic arthritis is justified.

The fourth member of the type is recognized by its steady, remorseless progression in spite of all treatment, by its stiff, shrunken joints, and its contractures. In their early stages the two forms cannot be distinguished. It is only the absolute failure of the one to respond to treatment, and its slow, steady progression, which enable us to distinguish it from the other.

Gout also may be considered in this connection. True gout is rare in this country, at least in the three widely separated parts of the country in which I have lived. It has a marked predilection for the metatarsophalangeal joint of the great toe, and the characteristic chalky deposits in the cartilages of the ear and the joints. The X-ray plate shows the punched out areas near the joint line.

The arthritis of gout is probably a traumatic arthritis caused by the deposition in the joint cartilage of crystals of biurate of soda. Its treatment is essentially within the domain of internal medicine, though the attacks of acute arthritis may perhaps be rendered less painful by rest.

PROGNOSIS.—The prognosis is on the whole good. In the early stages, however, on account of the difficulty or rather the impossibility of distinguishing this form from the hopeless progressive form, in the early stages, the prognosis must be guarded. Any multiple, progressive arthritis must be viewed with suspicion until its nature is known. As has been said, the bone and cartilage changes in this form usually are not great, and, as long as the disease is confined to the synovial membrane, there is no reason why complete recovery should not take place.

TREATMENT

This consists first and foremost in the removal of the cause. If the tonsils are suspected, they should be excised *in toto*. If no other focus be discovered, the tonsils, even if they appear normal, should be removed. If any evidences of infection be found in the deep urethra, treatment should be directed to that region. Sometimes irrigations and dilation, and prostatic massage will accomplish the purpose; often seminal vesiculotomy will be necessary. Tonsillectomy often is not followed by immediate improve-

ment. Indeed the operation is sometimes followed by an aggravation of the symptoms, and this aggravation is rather a favorable portent. A period of rest will be of advantage after the operation, in the severe cases even a week or two in bed.

After the removal of the focus, the joint, or joints, may return to normal, or to a condition approaching normal. If the symptoms continue, there are various measures which promise more or less relief. Chief among these perhaps is the deep intramuscular injection of a foreign proteid. The identity of the proteid is probably a matter of indifference. Antigonococcic serum or vaccine, typhoid vaccine, or horse serum may be tried. Baking, hydrotherapy, heliotherapy, and gentle massage have their advocates. Their rationale does not seem quite clear, but they may do good, nevertheless. If the pain be severe, immobilization may still it, but immobilization in this form of arthritis rarely is of much permanent benefit.

CHAPTER III

CHRONIC PROGRESSIVE MULTIPLE ARTHRITIS

THIS form differs from the preceding, as has been said, chiefly in its more or less steady progression, in its utter lack of response to treatment, and in the fact that nothing is known as to its cause. It is seen most frequently in young or middle aged women. The essentials of its pathology are the same as those of the preceding form.

The disease usually begins insidiously with pain and swelling in one or more joints. The joint stiffens. Then, one after another, the other joints become involved, until the patient becomes a hopeless, bed-ridden cripple. While there is no absolute rule of progression, the disease manifests a distinct tendency to symmetrical distribution. The acute symptoms, having lasted for awhile in a joint, may subside, leaving

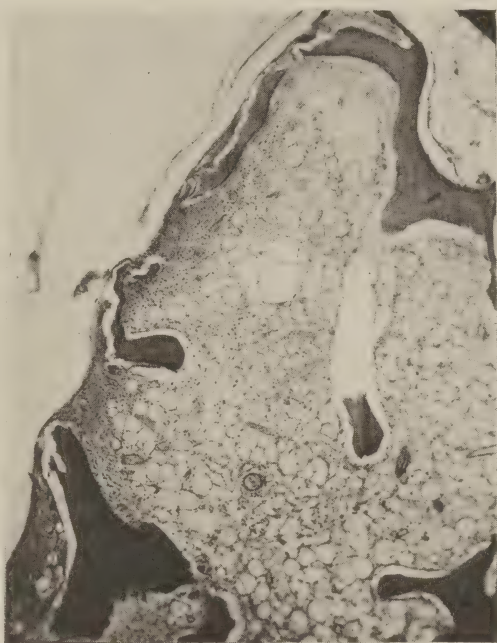


FIG. 111.—Low power photomicrograph of the bone at the articular surface, from a case of chronic arthritis of the first great type—the so-called rheumatoid arthritis. The specimen was removed at necropsy. Only the multiple lesions and the absence of tubercles in the marrow distinguish it from tuberculosis.

the joint more or less distorted and crippled. The joint thereafter is subject to other attacks, each one of which leaves it in worse condition. For weeks, perhaps, these attacks may be absent, and the patient and her friends think that she is on the mend; then the attacks recur.

The joints become ankylosed in semiflexion. The deformity is hard to prevent, and difficult to overcome when it has taken place. The wrists are in ulnar deviation. In America, indeed, this disease is often alluded to as "arthritis deformans," though it is quite distinct clinically and pathologically from the original arthritis deformans as described by the Germans. It is also sometimes called rheumatoid arthritis.

This form of arthritis has no set duration. For a long time, in the intermissions, the patients may be fairly comfortable, and their general condition may be fairly good. Sooner or later, some intercurrent disease, such as pneumonia, closes the scene. Sometimes a nephritis supervenes, and this fact justifies a supposition that an obscure infection is at the root of the trouble.

The prognosis is hopeless. I have been called to see a patient in the earliest stage of the disease, before there was any objective sign of an arthritis, except the merest suspicion of stiffness in the spine. I have watched her grow worse in spite of everything I could do, and I have seen her later as a clinic patient in the University hospital with all the resources of the staff at her service. Nothing that anyone could do was of any avail to check the onward march of the disease.

THE TREATMENT is symptomatic. The coal tar derivatives may check the pain, perhaps, in addition to external applications. Heat or cold may give relief. During the acute attacks splinting may be advisable. Morphine, on

account of its unfortunate after effects, must be employed sparingly, but sooner or later will probably be found necessary. Diet has no permanent effect upon the disease, and absolutely none in causing it, but of course the food should be carefully chosen and should be easily digestible. Fresh air and sunshine help to keep the patient in good general condition. Antisyphilitic treatment will always be tried and will invariably be found of no avail.

CHAPTER IV

STILL'S DISEASE

CHRONIC POLYARTHRITIS IN CHILDREN

IN 1897 Still¹ first drew attention to a form of polyarthritis in children, and published twelve cases of the disease. Since then several other observers have added reports of cases, but without clearing up the ætiology or the pathology. Evidently Still's disease belongs either in the third or fourth division of this great type of arthritis. Formerly it would have been classed distinctly in the fourth division, on account of its fatal termination, but the improvement, or recovery of several cases, and at least a rational guess as to the ætiology indicate that eventually it will be placed in the third, or at least, that many of the cases of it will be.

ÆTIOLOGY.—Nothing definite is known as to this, but the whole picture is that of a chronic infection. Reasoning from analogy we suspect the tonsil rather than the teeth.

PATHOLOGY.—I have not been able to find any account of an examination of the bone marrow, but the findings of several observers reveal that the changes in the synovial membrane and cartilage are typical of this type of arthritis; namely, the synovial proliferation, with the encroachment on the cartilage at its periphery, and the thinning and perforation of the cartilage. Rarefaction of the bone is shown by the X-ray, and in Whitman's case an enlargement of the proximal ends of the second row of metacarpals which might be mistaken for a spina ventosa. The skiagrams

¹ STILL, G. T.: "On a form of chronic joint disease in children." *Medico-Chirurgical Society Transactions*, London, 1897, lxxx, 47.

of several joints in Rosenfeld's case might be mistaken for tuberculosis in the absence of a history. Neither lipping nor spurring is present.

Hyperplasia of the lymph nodes, more or less general, is invariable. In one or two cases the mesenteric lymph nodes showed amyloid degeneration. The spleen is always enlarged, the liver often enlarged, and both may show amyloid degeneration. The same is true of the kidneys. Pericardial and pleural lesions are frequent, with or without valvular change. The thyroid may be enlarged, and exophthalmos may be present.

SYMPTOMATOLOGY.—Still's disease usually begins with chills, fever and sweating. At the onset, or some time afterward, the joint symptoms appear, with swelling, limitation of motion and pain. The larger joints are usually affected first, especially the knees, then, in much the same manner as in the preceding division, the other joints of the body become involved, one after the other, and more or less symmetrically, and as a rule to the accompaniment of constitutional disturbance. The sternoclavicular joint usually escapes. The same flexion deformities, contractures and swelling are observed as in the preceding class, even to the ulnar deviation of the hands. In contradistinction to the second great type of arthritis, the fingers in this disease, as in the other members of the type, show involvement of the proximal interphalangeal, rather than of the terminal joints.

The disease has its periods of acute exacerbation. In the intervals of the attacks, the patient may be fairly comfortable.

Various skin eruptions have been noted, and blood changes are characteristic; namely, leucocytosis, eosinophilia, and, sometimes, degeneration in the red cells.

These indicate extensive marrow disease. The lymph nodes are palpably enlarged, and usually increase in size during the acute exacerbations. The characteristic enlargement of the spleen can be made out, and often the enlargement of the liver.

The thyroid gland may be enlarged, and exophthalmos may be present. The urine often contains albumin.

Muscular atrophy is extreme and the shrinking of the muscles exaggerates the appearance of swelling in the joints.

PROGNOSIS.—The early cases reported all ended fatally. Recoveries have been reported in later cases, some of them, strangely enough, after an intercurrent infectious disease—scarlatina especially.

TREATMENT.—The first indication is to find the cause, if possible, and to remove it. The tonsils come first. They should be excised, and any other focus should be cleaned up. Even the middle ear and the sinuses should not be overlooked, if no improvement follow tonsillectomy. One patient showed improvement after the injection of a foreign protein. Palliative treatment may be advisable for the pain—splinting, heat, salicylates, etc. In the intervals, massage may be of benefit.

SECTION VII

THE SECOND GREAT TYPE OF CHRONIC
ARTHRITIS

CHAPTER I.

THE SECOND GREAT TYPE OF CHRONIC ARTHRITIS

SYNONYMS: Hypertrophic Arthritis, Degenerative Arthritis, Osteoarthritis, Arthritis Deformans, Metabolic Arthritis, Senile Arthritis.

ÆTIOLOGY

This is the mysterious type of arthritis, whose exact ætiology always has been in doubt. It is an ancient disease, as evidenced by its characteristic marks on bones of a great age. While its prevalence is greater in some regions than in others, its distribution is very wide, perhaps universal. It is essentially a disease of middle and of later life, and it never occurs in childhood. The term "arthritis deformans juvenilis" is a misnomer. I think that no case in a child ever has been published, which showed the lesions characteristic of the disease. The exact time of its incidence is sometimes hard to determine, but in 90 cases tabulated at the Stanford Clinics, the patients, when they presented themselves for treatment, were in the third decade of life in 2 cases, in the fourth in 12 cases, in the fifth in 26 cases, in the sixth in 32 cases, in the seventh in 15 cases, in the eighth in 3 cases. Almost two thirds of the patients were between 40 and 60 years of age. As the symptoms dated back some time before the patients were seen, it is evident that the onset of the disease is earlier than these statistics show. The age of the youngest patient was 28 years, of the oldest 60.

Various views have been advanced as to the prime cause. Most writers recognize a traumatic element in the causation, and some consider trauma the sole cause.

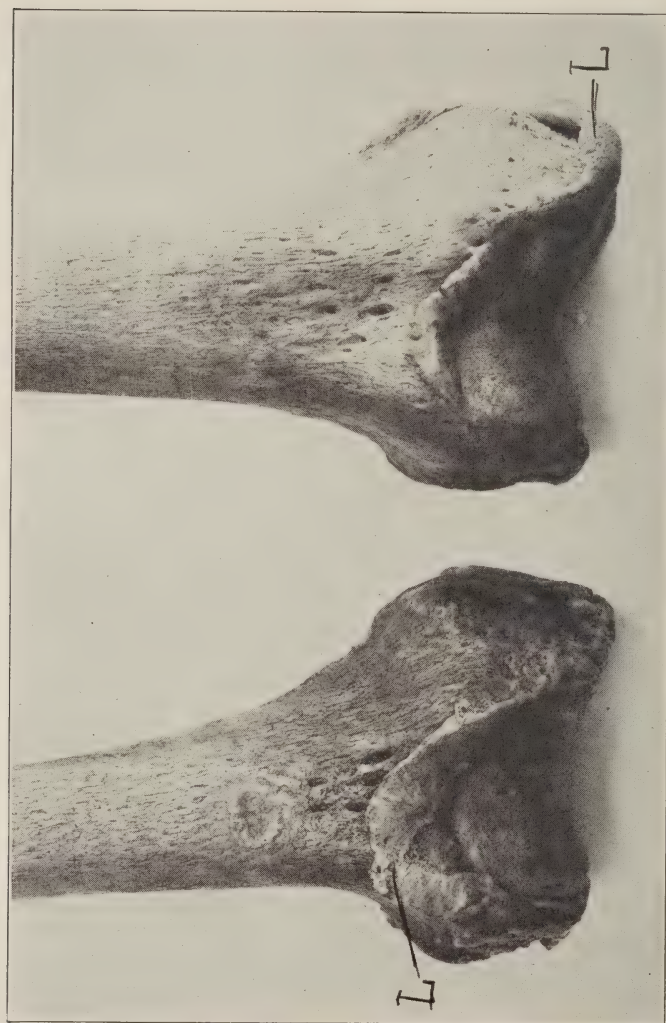


Fig. 112.—Distal end of two femora showing the typical lipping of the great second type of chronic arthritis.

Axhausen, as the result of laboratory experiment, maintained that the disease regularly followed injury to the articular cartilage.¹ Ely and Cowan reached conclusions diametrically opposite.² Chronic strain, whether due to laborious occupation, or to deformity elsewhere in the body, has been linked up in a causal relation, but the disease occurs in people in all ranks of life, and in the young it never follows dislocations, rotary lateral curvature, or even bow-legs and knock-knees.³

Many writers regard infection as the cause of the disease, but I have never been able to find reliable evidence of infection in any case. The evidence on this point was purely presumptive. Believing firmly that the infection was there, I have made unremitting but until recently vain efforts to find it. We must not permit our enthusiasm in these matters to influence our conclusions.

Exposure to cold and dampness, chilling of the surface, emotional and digestive disturbance, have all been held responsible, and there seems little doubt that these factors do influence the march of the disease, and aggravate the symptoms, but none of them is necessary to the incidence of the disease, and none of them is held to be its prime cause.

According to some, mysterious chemicals, floating in the blood, act directly on the bone and cartilage, and produce in them the characteristic changes. The terms "dyscrasia" and "diathesis," "rheumatic" or other, are employed

¹ AXHAUSEN, GEORG: "Ueber einfache; aseptische Knochen-und Knorpel-necrose, Chondritis dissecans and Arthritis deformans." *Arch f. klin, Chir.*, 1912, xcix, 519.

² ELY, LEONARD W., AND COWAN, JOHN FRANCIS: "Reaction of the tissues of the knee-joint of the rabbit to injury: Bone and Joint Studies I." Stanford University, Cal. Published by the University, 1916.

³ PATEK: "Static deformities as factors in the production of so-called hypertrophic arthritis." *J. orth. surg.*, 1921, iii, 324.

in this connection, and at present "faulty metabolism" is quite popular. Faulty metabolism, when analyzed, means simply, disease.

If one will carefully examine one's patients with this form of arthritis, one will find that practically all have

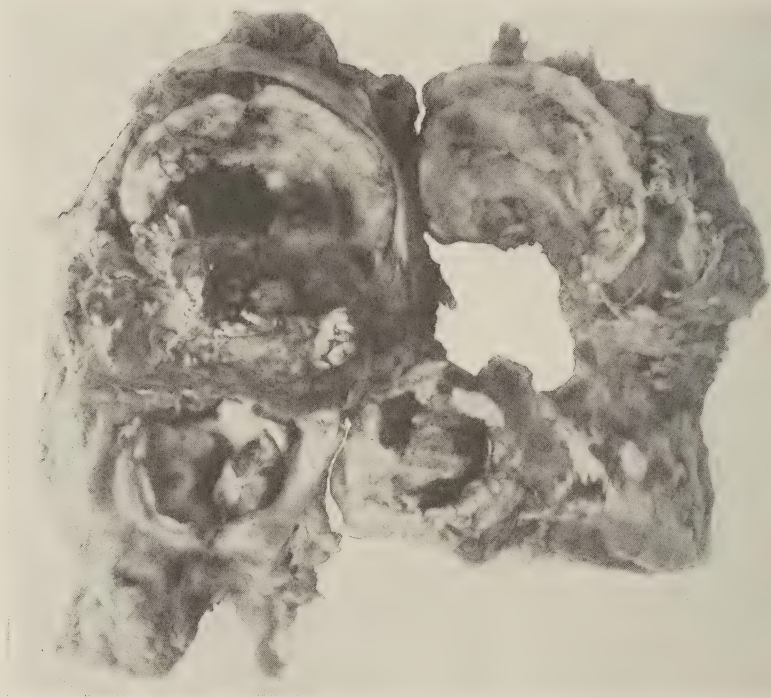


FIG. 113.—Photograph of a resected knee from a typical case of chronic arthritis of the great second type. Tibia on the left, condyles of the femur on the right.

one thing in common, namely, evidences of a chronic osteomyelitis about the roots of the teeth. I have found it in every one of my private patients. Two only of all my clinical patients, in whose history the condition of the teeth was noted, had sound teeth. The X-rays showed extensive rarefaction in the bones.

No direct proof of the causal relation of alveolar infec-

tion to the arthritis ever has been adduced, but the circumstantial evidence that a causal relation exists, is almost overwhelming. This hypothesis would explain the well known predilection of the disease for later life, and in earlier life only when the teeth are affected. It would explain also the influence of emotional and digestive disturbance, possibly also that of wet and cold, through the change in the secretions of the mouth. As I shall attempt to show, the primary changes about the joints are in the marrow in the vicinity. The changes in the cartilage and bone are secondary to them. Trauma acts by spraining a joint already damaged and distorted by the disease. A damaged machine is easily injured. Trauma is the cause of the subjective symptoms, not of the disease itself. On the other hand, trauma is probably the cause of the *arthritis* itself, that is, of the inflammation in the synovial membrane.

In my earlier work I considered the alveolar osteomyelitis as the direct cause of the disease, but the two patients with sound teeth negatived this theory, as well as the inherent improbability of it. The most probable hypothesis is that the offending organism is domiciled in the intestinal tract, and is ordinarily harmless as far as the joints are concerned, unless it have a port of entry. The alveolar osteomyelitis furnishes this in the vast majority of cases. The organism may be a protozoön, and a group of us are working to find out its identity. The necrosis in the bone points to the *amœba histolytica* as the culprit.⁴

⁴Since the above was written we have found the *amœba* in the bone marrow of one case, and have reason to believe that we shall find it in others. If the finding be confirmed, the subject will be put on a scientific basis.

ELY, LEONARD W., ET AL.: "The *amœba* as the cause of the second great type of chronic arthritis." *Cal. State Med. Jour.*, 1922, xx, No. 2.



FIG. 114.—Chronic arthritis of the great second type. Note spurring on the condyle and tuberosity, and the cavity at C. The presence of cavities in the bones was demonstrated at operation in this case.

PATHOLOGY

The gross pathological changes in this type of arthritis have had extensive mention, and are well known to many who are called on to treat bones and joints. Very few

have attempted to work out the pathological histology Nichols and Richardson's work is a classic in this line.

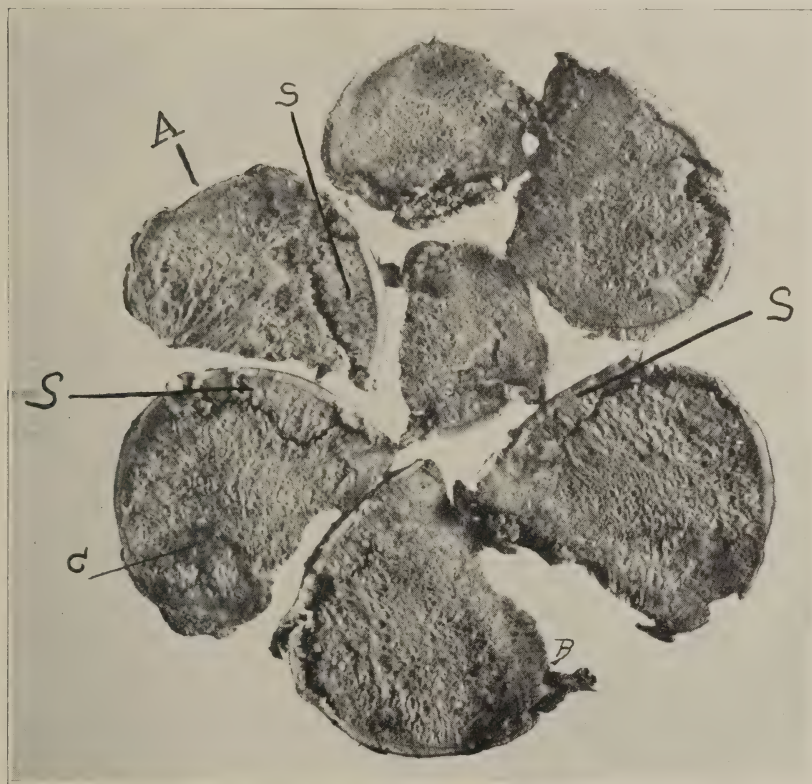


FIG. 115.—Sections of head of femur from a case of second type arthritis. S, sequestrum.

When one examines a specimen of this form of arthritis in the laboratory, three things are prominent in the naked eye inspection:

1. Thickening, reduplication and villous proliferation in the synovial membrane.

2. Partial or complete disappearance of the articular cartilages, with a dense, hard, polished, eburnated (ivory-

⁵ NICHOLS, E. H., AND RICHARDSON, T. L.: "Arthritis deformans." *Jour. Med. Research*, 1909, xxi, 149.

like) condition of the subjacent bone. This bone is often grooved in the line of joint motion.

3. Masses of bone and cartilage built up at the margin of the articular cartilage, at the line of attachment of the capsule. It is these masses of new bone which show in the X-ray plate, and enable one to recognize the disease clinically, and are responsible for most of the names that have been bestowed upon it. Pieces of bone and cartilage may



FIG. 116.—Photograph of a stained slide from section A of the preceding.
S—Sequestrum. L—Lipping.

lie loose in the joints, or may be attached to the bone end or to the synovial membrane.

Upon sectioning the bone one finds larger and smaller places in which the bone tissue is lacking.⁶ These spaces may be filled with fibrous marrow, or they may be the seat of cysts, large and small. Sometimes fibrous tissue and cysts are intermingled, the proportion of each varying in different sections.⁷ Small pieces of dead bone may be

⁶ ELY, LEONARD W.: "A study of 100 dry bones sawn in the laboratory; Bone and Joint Studies I." Stanford University, Cal., published by the University, 1916.

⁷ ELY, LEONARD W.: "The great second type of chronic arthritis." *Arch. of Surg.*, 1920, i, 158

found in the fibrous tissue. Occasionally a large part of the end of the bone may be nothing but an aseptic sequest-

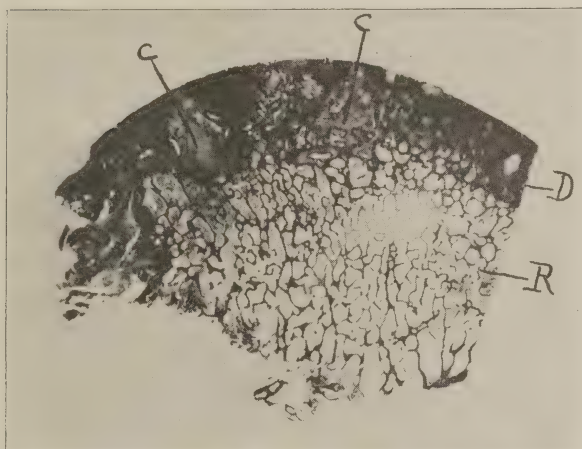


FIG. 117.—Photographs of stained slides from sections taken a short distance from each other from the head of the femur in a case of second type arthritis. Note cavities at C, the layer of dense bone, D, at the articular surface of the bone and the rather open meshed bone beneath it.

rum. *The aseptic necrosis is probably the primary morbid change.*

The ridges of bone at the margin of the joint cartilage, the lipping or border exostoses, as they are called, are seen

to consist of bone usually covered by cartilage, and raised above the level of the articular surface of the bone. New bone evidently has been laid down also in the marrow in the immediate neighborhood of the joint, for the bone tissue here is wont to be much denser than normal. Deeper

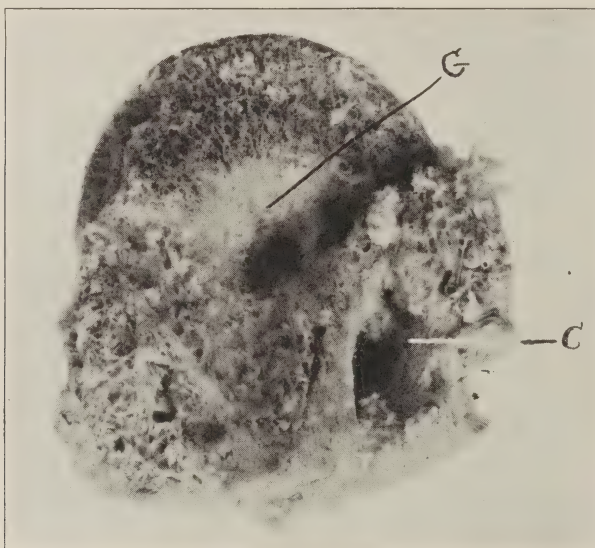


FIG. 118.—Section through the neck of the femur from a case of second type arthritis. C, cavity. The cavity marked G, is an artifact caused by gouging out the bone for culture purposes.

in, it is more open meshed than is normal bone in the same region.

HISTOLOGY

Besides all the bone patent to naked eye inspection, new bone can be seen forming in the marrow, both through the medium of cartilage and directly out of fibrous tissue. The marrow is mostly fibrous and fatty. Here and there are areas in which all bone is lacking. Its place is taken by cysts and fibrous tissue in varying proportions. Sometimes in the fibrous tissue small dead trabeculae can be dis-

tinguished. I have one specimen of a femoral head, which consists of little else than a large sequestrum, but there is nothing in my specimens to indicate that arterial disease is the prime cause of the necrosis. Elsewhere the bone trabeculae are usually thicker and denser than normal.

The joint cartilage is irregular both in its thickness and in its structure. In places it is thicker than normal, in

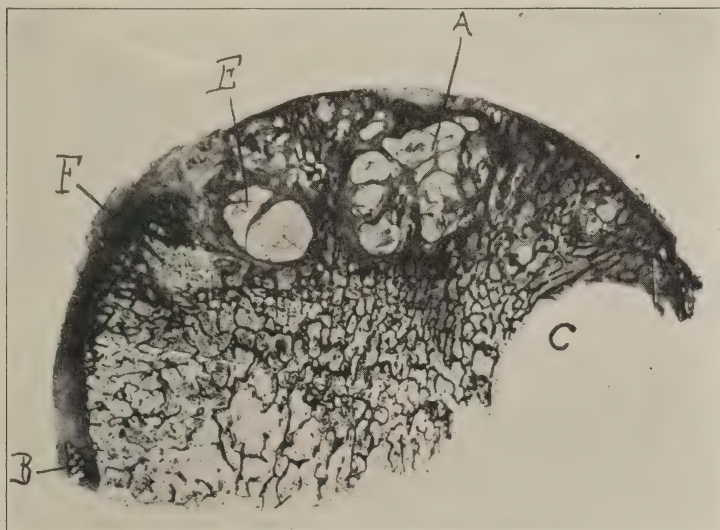


FIG. 119.—Photograph of stained slide of a section of the head of the femur from a case of second type arthritis. A, E, F, C, cavities in the head of the bone. B, bone formation in the articular cartilage.

places it may be absent altogether. Its structure is best described as bizarre. It sometimes presents a fantastic appearance. Generally it is fibrillated. Often its cells are swollen and distorted, often many of them have disappeared. Its surface may have a peculiar tattered look. Calcification of the cartilage to a greater or less extent is common.

The marrow shows areas infiltrated by lymphocytes, and among the cells many plasma cells can be made out.

The synovial membrane is thickened and villous. The villi, while they may show some cellular proliferation at their surface, have not the cellular, "lymphoid" structure so common in the first type of arthritis, but consist usually of a loose-meshed reticulum of fibrous tissue containing considerable fat. Bone and cartilage formation is said to take place in the villi. Lymphocytic infiltration takes place in the synovial membrane as well as in the marrow.

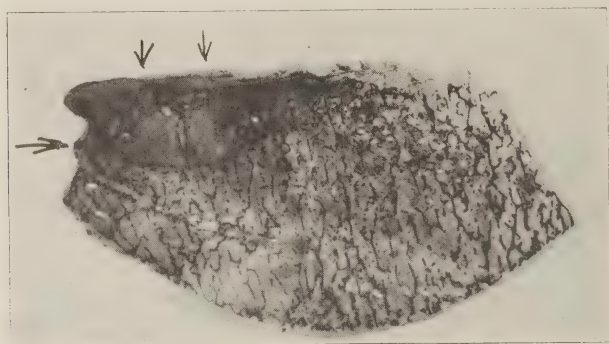


FIG. 120.—Photograph of stained slide from a case of second type arthritis of the knee. Articular surface above. The arrows point to the necrotic areas in the bone.

The absence of the cartilage, with the resulting exposure of the underlying bone, causes a grating and creaking of the joint when it is moved, and the fact has given rise to the opinion that the joint was "dry." It is not dry, but usually contains an excess of serous fluid.

In the spine, new bone formation takes place in the anterior common ligament, occasionally welding the vertebræ together. The usual production of bone consists of lipping of the vertebral bodies, as in other bones. In the other joints of the body union of the articulating bones, whether by bone or by fibrous tissue, practically never takes place. The limitation of motion is caused by the roughening and distortion of the bone ends. The joint is

damaged as a machine for locomotion. The cartilage, once worn off, never re-forms, and the new bone formation is never absorbed.

When the disease occurs in the hip, it sometimes passes under the name of "*morbus coxae senilis*." New bone is deposited not only about the femoral head and the acetabulum but also on the great trochanter. The head of the

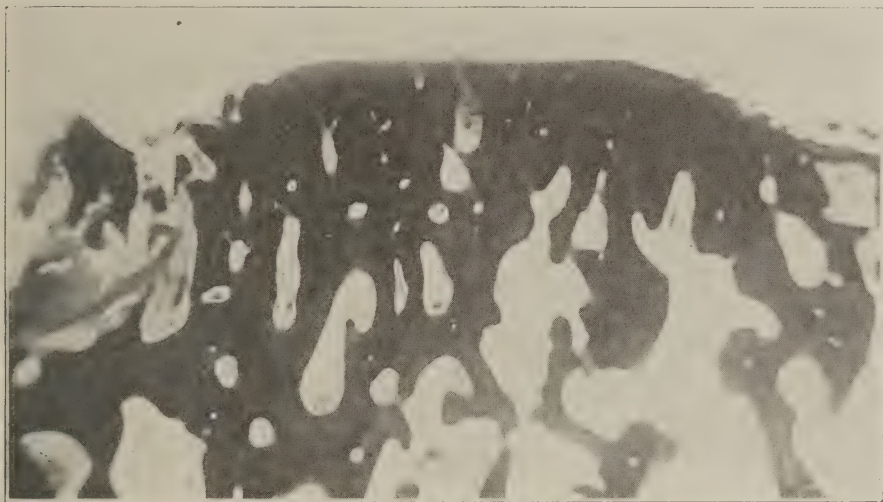


FIG. 121.—Eburnated bone at the articular surface in a case of second type arthritis. Low power photomicrograph.

femur often becomes flattened, "mushroomed," as it is called, and ceases to fit in the acetabulum. Actual dislocation however probably never takes place. The Röntgen film taken immediately after a fracture of the hip in the aged often shows the characteristic changes of this type of arthritis. The fracture does not cause the arthritis, nor does the arthritis cause the fracture, but both are caused by the same thing, namely, rarefaction in the head and neck of the femur. I think this rarefaction, but not necessarily

the arthritis, will always be found in the peculiar fracture of the femoral neck occurring in the aged.

In the fingers, the disease affects by preference, the terminal interphalangeal joints, in contradistinction to the first type of arthritis, which affects usually the proximal interphalangeal and the metacarpophalangeal. The terminal phalanges become semiflexed, and slightly deflected

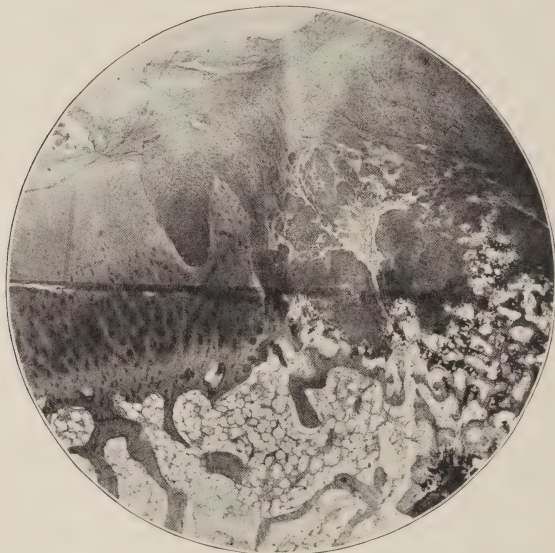


FIG. 122.—Low power photomicrograph of articular cartilage from a case of second type arthritis. Note its bizarre appearance and the marked evidences of calcification.

laterally. The bone production about the joint line gives rise to characteristic deformities, popularly known as Heberden's nodes. These are sometimes considered as a manifestation of gout, but they have nothing whatever to do with that disease. Study of the X-ray pictures of these finger joints teaches us that the bone production is never as much as it appears clinically. Rarefaction and bone destruction dominate the picture. Unless this fact be borne in mind, one may err in the diagnosis.

This type of arthritis is practically always multiarticular in its manifestations in the spine and in the fingers. When it occurs elsewhere, the symptoms may be confined to one joint, but if other joints be radiographed, the characteristic lesions will often be found in them also. The essential primary change, as we have seen, is probably in the marrow, and this change may exist for a long time, and

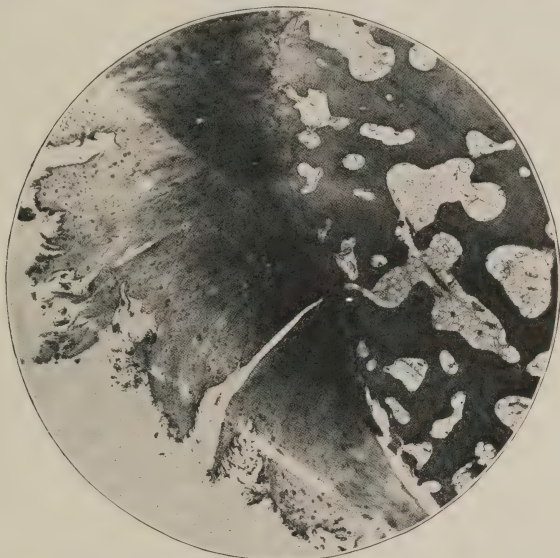


FIG. 123.—Typical appearance of cartilage from a second type arthritis of the hip. Low power photomicrograph. Note calcification, fibrillation and "tattering."

may be widespread before it produces any symptoms in the joints. We regard the disease as an arthritis because it manifests its presence by joint symptoms, but fundamentally it is perhaps the same process as that at the bottom of certain diseases of the shafts, described in another section.—Paget's deforming osteomyelitis and osteomyelitis fibrosa.

Radiographs of intraarticular fractures in middle aged and elderly persons indicate that much of the stiffness

following the fracture is due to the opening up of an old focus in the diseased marrow. Indeed it may well be that Colles' fracture, for instance, as well as fracture of the neck of the femur, owes its frequency to a preexisting rarefaction in the bone.

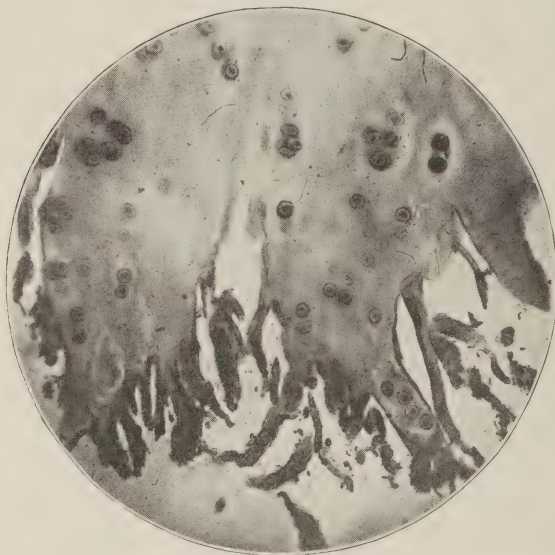


FIG. 124.—Rather high power photomicrograph of some of the cartilage tatters shown in figure 123.

SYMPTOMATOLOGY

The symptoms are those of a low grade chronic arthritis—pain, moderate swelling, stiffness, limitation of motion, disability, deformity, etc.

The pain is rarely severe except in disease of the hip, and is almost always lessened by heat: Hence the tendency of elderly persons with "chronic rheumatism" to hug the fire. The joints, being mechanically damaged, are subject to strains and sprains. These are followed by an exacerbation of the symptoms, and in the spine are called "neuritis," "lumbago" or "sciatica." The pain is usually

aggravated by cold and dampness. The joints of persons with this form of arthritis often constitute an excellent barometer.

Errors of diet also increase the pain; lobster, strawberries and tomatoes will often bring on an attack.



FIG. 125.—Cellular infiltration in the marrow from a case of second type arthritis. Low power photomicrograph.

Patients therefore think that they are “gouty.” Any idiosyncrasy of diet may have its effect. Sometimes the pain is worse on rising, wearing away as the joint is used, but overuse increases it. The joint creaks and grates, sometimes audibly, almost always palpably. If the pain is severe, as in disease of the hip, or of the spine after strain, muscular spasm may be marked, otherwise it is not to be

expected. The spine may be held perfectly rigid during an acute attack.

The swelling is usually moderate, and is due to the thickened synovial membrane, to the fluid in the joint,

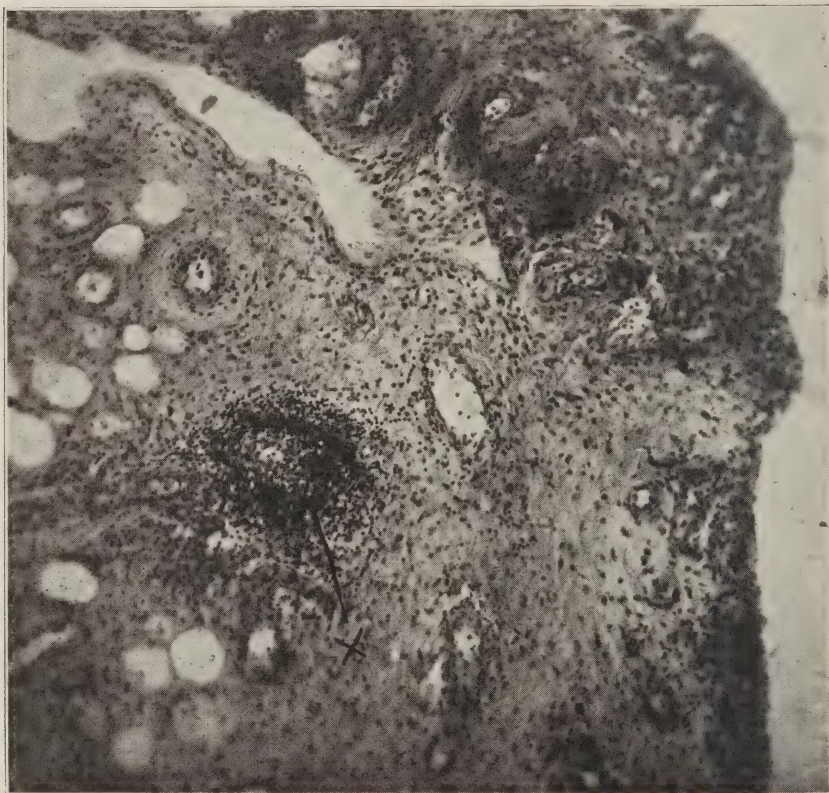


FIG. 126.—Low power photomicrograph of a section of the synovial membrane from a case of second type arthritis. The membrane presents an appearance quite different from the one peculiar to the first type. Note the fatty and loose-meshed fibrous tissue, and the area of cellular infiltration at X.

and to the new bone. In the knee it is plainly evident, in the spine or hip it cannot be made out. It is not constant, but varies with the progress of the disease.

The characteristic deformity is slight flexion. Extreme degrees of flexion, such as occur in the first type of arthritis, are not met with in this type. In the spine a rounded

kyphosis, rarely approaching angularity, is fairly standard. Again, the contour of the spine may be perfectly normal with extensive bone changes, or the normal curves may be simply slightly changed. Often a lateral curve is the most prominent feature.

The hip, besides being in semiflexion, may be in adduction or abduction. External rotation is more frequent than internal. When the tarsal joints are involved, the feet are abducted, flattened and stiff, giving the peculiar gait, so beautifully seen in hotel waiters. These are the stiff, painful flat feet that have caused so much discussion. When the X-rays show the characteristic bone changes, the arthritis is thought to be due to the flat feet, whereas the flat, rigid feet are the result of the primary arthritis. An element of equinus is also present in the deformity, only to be distinguished when the foot is brought into adduction.

Limitation of motion varies according to the joint involved, and also with the exacerbations of the disease. In spinal disease, with bony ankylosis, the limitation is absolute in the region affected. In the spine, in the feet, and in the hip, the limitation may be marked. Two elements enter its causation, namely, muscular spasm, and change in the articular surface of the bone. The second element is a purely mechanical one. In the spine, flexion may be free and extension limited, or the reverse. However, the X-rays may show extensive changes in the vertebrae in the face of a perfectly flexible spine. Rotation, adduction, and especially abduction, are all limited in disease of the hip. A peculiar diagnostic sign in this form of arthritis is, that when flexion is forced, the hip goes into abduction.

In the other joints the amount of limitation of motion is roughly proportional to the extent of the bone changes.

Two physical signs merit attention in this connection. In sacro-iliac arthritis, a Kernig sign is present on the affected side. It may be present also in lumbar arthritis. An Ely sign is often present in lumbar arthritis.⁸

COURSE OF THE DISEASE.—This type of arthritis has no set course. It is essentially chronic, and is more or

less progressive in a joint until its cause is removed. Then it usually becomes quiescent. Its course is influenced by trauma, and by hard usage; hence partly, the preponderance of patients with laborious occupation, though it may be said on the other hand that people in the poorer classes do not take care of their teeth.

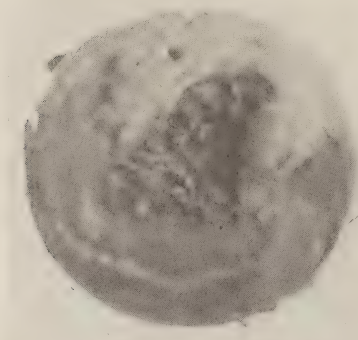


FIG. 127.—Articular surface of head of femur resected eighteen months after fracture of the femoral neck in a patient seventy-three years of age. Note the second type changes.

The disease occurs in persons of all ranks, but well-to-do patients, who are not driven to laborious occupations, usually escape its more painful manifestations.

Diet and emotional disturbance also influence the course, as do exposure to dampness and cold. Here we have the whole clinical picture of the disease. Poor people form the majority of the patients, probably because there are more poor people than rich. They neglect their teeth, but usually have them extracted early, whereas the more prosperous person, when his teeth decay, resorts to elab-

⁸ The patient is laid prone upon the table. If his pelvis rise from the table when the surgeon flexes his knee, he is said to have a positive Ely sign. The phenomenon is probably caused by the pull forward on the rigid spine by the rectus muscle.



FIG. 128.—Chronic arthritis of the great second type; typical changes in the fingers—Heberden's nodes.

orate and expensive dentistry. His mouth is filled with dead teeth, crowned, capped and filled. The X-rays show the presence of abscesses about the roots of these teeth, but they usually occasion no pain, and therefore the patient is naturally loath to have the teeth extracted. The poor man must work hard, must eat poorly prepared food, and is exposed to inclement weather. He is the one therefore who suffers most from the severe manifestations of this form of arthritis. The prosperous person need do no hard physical work, and can keep his body protected from the rigors of climate. His food is properly prepared, and he can eat what he has learned by experience, will agree with him. If his joints become troublesome, he goes to some health resort where he is kept quiet, where his diet is carefully regulated, and where heat in some form is applied to the surface of his body.

DIAGNOSIS

This rarely occasions much difficulty. It can be made with reasonable accuracy by clinical examination, but it can be established beyond peradventure by the X-rays. If the bones show the typical lipping and spurring about the lines of attachment of the capsule, the arthritis belongs in this type. In obscure "neuritides," "sciaticas" and "lumbagos" the spine should be radiographed.

In a Charcot joint, there may be an irregular production of new bone, but it is not in the form of lipping and spurring, and it does not follow the line of capsular attachment. Marked disorganization of the joint is present, and, instead of limitation of motion, supermobility. The Charcot joint is painless, and the patient shows signs of tabes.

PROGNOSIS

The new bone built up around the joint is a fixture, and shows no tendency to absorption. Its presence indicates extensive changes in the cartilage and in the marrow, and its removal therefore is not followed by cure. The synovial hypertrophy also is the indirect result of the changes in the bone and cartilage, and there is no object in trimming off its villi and its redundancies.

If the cause of the disease can be removed before great changes have taken place in the bone and cartilage, the pain may disappear, and the joint may return to a state approaching normal. Without appropriate treatment the arthritis has a tendency slowly to grow worse.

This form of arthritis carries with it no particular danger to life, unless the spine be extensively involved.

In that case respiration may suffer interference, and the patient may be liable to pulmonary disease, though rarely to tuberculosis. For some unexplained reason, the type

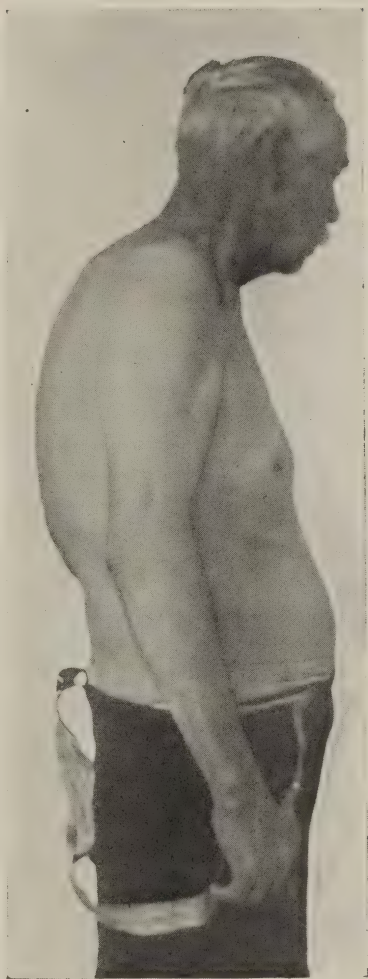


FIG. 129.—Chronic arthritis of the great second type involving the thoracic spine. Note the typical bowing involving a large part of the spine.

of person that is afflicted with this form of arthritis usually does not suffer from tuberculosis.

TREATMENT

No recognized standard treatment exists for this disease. The differentiation between the two great types of



FIG. 130.—The same patient as shown in the preceding figure. In spite of extensive disease no real muscular spasm is present.

chronic arthritis, has never been sharply made by most writers, and the treatment recommended for one, usually is the same as that recommended for the other. The two are entirely different, as has been shown in the preceding pages, and should be handled along different lines.

As I view the disease, the essential pathological feature is the aseptic necrosis in the bone near the joint line, and the almost invariable cause is the chronic osteomyelitis in the alveolar processes of the jaws. I recognize that the

bone and cartilage changes are permanent, and that when the architecture of the joint has once been seriously damaged by this disease, it can never be restored. The problem of making cartilage grow again over the end of the bone is strikingly like that of making hair grow again on a bald head. Therefore the best I can do when a patient presents himself for treatment, is to stop the disease where it is, with the idea that, when I remove the cause, provided the bone changes are not excessive, the changes in the soft tissues will disappear and the symptoms will subside.

The first indication is to remove every focus of infection from about the roots of the teeth. Its presence usually, but not always, can be determined with the X-rays. Dead teeth are always to be viewed with suspicion, even when the radiogram does not show any rarefaction about their roots. They are foreign bodies, and it is doubtful if the bone is ever healthy in their immediate vicinity. Sometimes an old, buried root is discovered. It must be dug out. The wisdom of the so-called "surgical" removal of teeth is at present being urged, that is, the necessity of chiselling away the alveolar processes. Personally I do not think it is necessary.

The removal of the focus in the mild cases may be all the treatment necessary. The symptoms promptly disappear and so does the patient. In the majority of cases however some discomfort and stiffness remain. They can be made less by physical therapy. Heat in some form is almost always grateful to patients with this type of arthritis. Baking, ironing out with a hot flat-iron over several layers of blanket, hydrotherapy will all be found useful. The so-called Bier treatment, passive hyperæmia with the employment of the Esmarch bandage, seems to relieve the

pain in the joints of the extremities. The bandage should be applied for an hour or so daily, just tight enough to turn the extremity dusky red and warm, not tight enough to turn it blue and cold.

A search for parasites in the stools should be made, and if they be found, treatment for their removal should be instituted.

For some reason, the intramuscular injection of a foreign protein seems to act favorably in some cases. The identity of the proteid is not important. Typhoid vaccine will serve, or antigenococcus serum or vaccine.

We recognize the subsidiary rôle of cold, dampness, strain, emotional disturbance and diet. We keep the patients warm, and warn them against exposure, recommending a residence in a suitable climate if possible.⁹ We guard against strain, and regulate the diet according to the patient's idiosyncrasies. Most persons reaching adult life know perfectly well what they may safely eat, and what they may not, but few have the requisite will power to prescribe a diet for themselves. It is a cynical way to look at it, but the patient's idiosyncrasies can be extracted from him, and then a suitable diet can be prescribed with the requisite amount of camouflage. In these chronic diseases the psychological element must not be neglected. All indigestible food is *tabu*. Generally, shell fish, strawberries, tomatoes and red wines are objectionable. Sugar as a rule should be ingested sparingly. Red meats do not agree with some people, fish with others. Milk, as an article of diet for an adult, is probably greatly overrated. Tea, coffee and tobacco, are all poisons that agree well with most persons.

⁹ Climate is not everything in the treatment of this disease. The climate of California is renowned, but this type of arthritis is prevalent in California.

My experience teaches me that the treatment outlined above is more rational and more satisfactory than any other, but the experience of other observers, many of them of deservedly high reputation, leads them to entirely different conclusions. Like any disease, whose ætiology and pathology have not been definitely established, this type of arthritis is treated in most diverse ways.

Pemberton¹⁰ does not differentiate sharply between the two great types. He considers chronic arthritis generally to be caused by metabolic disturbance, and strongly urges its treatment by measured diet. The chief point in his thesis, is the harmfulness of the carbohydrates, and he reduces their ingestion to a minimum.

Many observers fail to make the sharp distinction between the two great types. Many observers make the distinction, but think that infection in the tonsil, in the jaws or in the deep urethra can cause either type of arthritis indiscriminately, and advise the removal of the focus wherever it is found, for either type of arthritis. This view, I think, is held by most men whose opinion is of value. The exclusive rôle of alveolar infection is original with me, and has not been accepted by anyone except by some of my immediate associates.

All manner of drugs, external applications and physical therapy, have been recommended. Salicylates often will relieve the pain. Strange to say, immobilization is not well tolerated by most patients, but in spinal arthritis a Taylor brace protects from strain, and sometimes makes the pain less. In sacroiliac arthritis a Goldthwait belt answers the same purpose.

¹⁰ PEMBERTON, RALPH: "The nature of arthritis and rheumatoid conditions." *J. A. M. A.*, lxxv, 1759.

PEMBERTON, RALPH, AND ROBERTSON, J. W.: "Studies on arthritis in the army, etc." *Arch. Int. Med.*, 1920, xxv, 231.

In tarsal arthritis, with accompanying rigid flat foot, Whitman's treatment is probably the best. Under general anæsthesia, Whitman wrenches the foot around into strong inversion and dorsal extension, pads it well, and puts it up in plaster of Paris for about six weeks. At the end of that time he removes the plaster, takes a cast of the foot, and puts the foot up in plaster again for a couple of weeks longer. Meanwhile he has a Whitman brace fashioned from the cast, and has the patient's shoe raised a quarter of an inch on the inside. When the second plaster dressing is removed, active and passive movements are started, and the patient wears the brace and the built-up shoe. The result of this treatment is usually excellent.

This type of arthritis in the hip is sometimes very painful, and the pain may persist in spite of all the ordinary methods of treatment. In such case resection offers the best way out. The head of the bone should be removed, preferably through the Sprengel incision, the trochanter should be thrust into the acetabulum, and the hip should be immobilized in a long plaster of Paris spica for a month or two. Probably the pain which often follows a fracture of the femoral neck, in the elderly, is due to this form of arthritis. Resection here also will be found satisfactory.

PRACTICAL CONSIDERATIONS

In the preceding pages, the investigator should find much that will help to a comprehension of the subject of chronic arthritis, and to a further advance of knowledge. For the general practitioner, who is called on almost daily to treat cases of chronic arthritis, a few practical hints may be in order.

In the first place, one should lay down a hard and fast rule, in the absence of distinct motor symptoms, never to make a diagnosis of a neuritis or neuralgia of the extremities, until a thorough examination, both clinical and skiagraphic, has excluded the presence of any lesion of the spine. Possibly an isolated peripheral neuritis may exist without motor symptoms, but it is improbable in the highest degree. Sciatica, lumbago, brachial neuritis and intercostal neuralgia usually can be traced back to a lesion of the spinal joints or of the cord itself.

The patient should be stripped invariably. A woman may be draped with a sheet for the examination. Any change in the contour of the spine should be noted, and then limitation of motion should be sought in any region of the column. A lesion of the sacro-iliac joint gives a positive Kernig sign, as a rule.

An inflammation of any joint of the extremities is usually easily detected, if only the patient's clothes are removed, and if the possibility of referred pain is kept in mind, especially of pain in the knee with hip joint disease. The joint is usually swollen, and almost invariably is limited in its motion. The latter is the chief diagnostic sign.

Having determined the presence of an arthritis, the next step is to put it in its correct class or type. This can usually be done by the clinical examination, and can always be done with the aid of the Röntgen rays. The question has been dealt with at length in the preceding pages.

If the arthritis is of the second great type, its cause is to be sought in the alveolar processes of the jaws, and any infection there is to be removed as the first step in the treatment. The joints must be protected from strain, and kept warm, and the diet must be regulated. A search should be made for parasites in the stools.

When the arthritis is of the first great type, the question is not always so simple. It can be caused by any one of a number of different agents, but the chief ones are the tubercle bacillus, the treponema pallidum, and the diplostreptococcus domiciled in the tonsil, or in the deep urethra of the male. An absolute clinical differentiation of these three is impossible, but the more carefully one searches the history and examines the patient, the less frequently will one err.

Tuberculosis may occur at any age, but a persistent single arthritis in the young is usually tuberculosis. Tuberculosis is slow in its onset and rarely involves more than one joint. If the bone is involved, the disease is wont to be very painful, and to be accompanied by marked muscular spasm and atrophy. Cold abscess practically excludes everything else except coccidioidal granuloma.

Syphilis may be single or multiple in its joint manifestations. It may be painless, but often is quite painful. The bone may be badly damaged in the immediate neighborhood of the joint, without interference with joint function. Glandular enlargement is frequent, and other evidences of syphilis, especially new periosteal bone formation. When the gumma breaks down, and when a sinus forms, the mouth of the sinus is wont to be dirty, dark red, undermined, and sluggish looking, whereas that of tuberculosis is pale, puffy, and pouting.

Diplostreptococcic arthritis, though sometimes single, is more often multiple. The joint may be swollen or shrunk, full of fluid, or a mass of adhesions. If the habitat of the organism is in the deep urethra, the arthritis may have supervened on an acute gonococcic infection, or it may be chronic from the start. When all is said, we usually make the diagnosis on probabilities. Involvement

of the finger joints speaks for the tonsil, as does a certain tendency to symmetry. Pain under the heels speaks for the deep urethra. If examination shows disease of the seminal vesicles or prostate, and pus in the tonsillar crypts, naturally we should first turn our attention to the urethral lesion, for a patient with detritus in the tonsil may be in health, but one with a deep urethral lesion never is. On the other hand a patient with a lesion in the deep urethra, may have an arthritis caused by tonsillar infection, and if his arthritis did not recover after treatment of the former, then we should remove his tonsils. We do not stop until we have done our best to remove every possible focus of infection.

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SECTION VIII

**ARTHRITIS CAUSED BY DEVELOPMENTAL
ABNORMALITIES**

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CHAPTER I

LEGG'S DISEASE

THERE are three diseases or conditions of the joints of the lower extremity, one in the hip, one in the knee, and one in the foot, whose exact nature never has been established, but which present marked similarity in more than one respect. Each one comes at a certain age, each affects boys more than girls, and all three are generally considered to be caused by trauma, though the infectious origin of one is at present being urged. Each causes an arthritis. The three are Legg's disease, the "essential" joint mouse, and Koehler's disease.

Legg's disease affects the epiphysis of the femoral head, and gives symptoms between the ages of five and ten, when the bone nucleus is at a certain state of development. The typical joint mouse forms in the medial condyle of the femur of adolescents, and gives symptoms just before the union of the lower femoral epiphysis with the shaft; Koehler's disease always affects the navicular bone of the foot, gives symptoms between the ages of three and nine, and causes no trouble when ossification of the navicular is finished. For these reasons, although the evidence is by no means conclusive, the grouping of the three lesions is justified.

On the hypothesis stated, the developmental abnormality may exist indefinitely without causing any symptoms. The joint however, considered as a machine, is defective, and is easily damaged. A slight injury, which, in a normal joint would occasion no trouble, here starts up an arthritis. In other words the arthritis occurring with these deformities, is to be considered as a traumatic arthritis.

LEGG'S DISEASE

PERTHES' DISEASE; OSTEOCHONDRITIS DEFORMANS
JUVENILIS; ARTHRITIS DEFORMANS JUVENILIS

This disease was described almost simultaneously by Legg of Boston, by Calvé of France and by Perthes of Leipzig, Legg antedating Calvé by about five months. Perthes followed Calvé in three months. It consists of a peculiar segmentation and change in shape of the femoral head.

ÆTIOLOGY.—Nothing definite is known as to this. The disease is generally considered to be caused by an infection. In two cases, a growth of staphylococcus is said to have been recovered from a soft area in the neck of the femur. Boys are more often affected than girls in about the proportion of 4-1. The typical changes of the disease are often observed in the Röntgen picture of congenital dislocations of the hip which have been reduced. They have been discovered accidentally also on several occasions in the sound hip of patients who have been operated upon for congenital dislocation. Like most joint diseases, this has been ascribed to trauma and to syphilis. Until the publication of Legg's paper, it was mistaken for tuberculosis, and probably the vogue of certain methods of treating hip tuberculosis in the past, has been due to this error.¹

The things which speak for the view that Legg's disease is essentially caused by a congenital anomaly of development of the proximal bone nucleus of the femur, are the following:

1. The condition is observed almost invariably at a certain period of growth. Practically all the patients are between the ages of five and ten.

¹The writer has been guilty of this error. In his book on joint tuberculosis appears a Röntgen picture of a case of cured hip tuberculosis. In the light of increased knowledge this is evidently a case of Legg's disease.

2. The typical deformity may exist indefinitely without symptoms, and may be discovered accidentally.

3. The condition is frequently bilateral.

4. It is seen often with congenital dislocation of the hip, not only in the affected hip after reduction, but also on the sound side.

5. An infection in the epiphysis severe enough to produce the bone changes which actually occur, would sometimes undoubtedly break into the joint and cause an infectious arthritis. No case of actual suppuration is on record.

6. When the case is properly treated, the bone slowly forms in the epiphysis, and a cure regularly results in about a year, with little deformity or with none at all. Without treatment, the deformity slowly increases, and terminates in a characteristic condition. The details of the argument can be supplied from a study of the symptomatology and of the scant pathological findings. If we faced an infection, the pathological material at our disposal would be far richer than it is.

PATHOLOGY.—As to this we know very little. Two operators claim to have recovered at operation a pure culture of staphylococcus from the marrow of the neck of the femur. One of these cases showed a “necrotic” area, the other a “grayish” condition of the marrow. Perthes excised a piece of synovial membrane and a piece of the femur head from one patient. The synovial membrane was normal. The bone showed irregular islands of cartilage in the marrow, near the articular cartilage. Neither the marrow nor the synovial membrane showed any sign of inflammation.

Phemister operated upon one case. The joint was full of a slightly turbid, straw-colored fluid, and the synovial membrane was villous. An excised tag of the membrane

showed "hyperplastic connective tissue elements with a few areas of round cell infiltration and a thickened synovial covering rich in nuclei." He found a cavity in the epiphy-

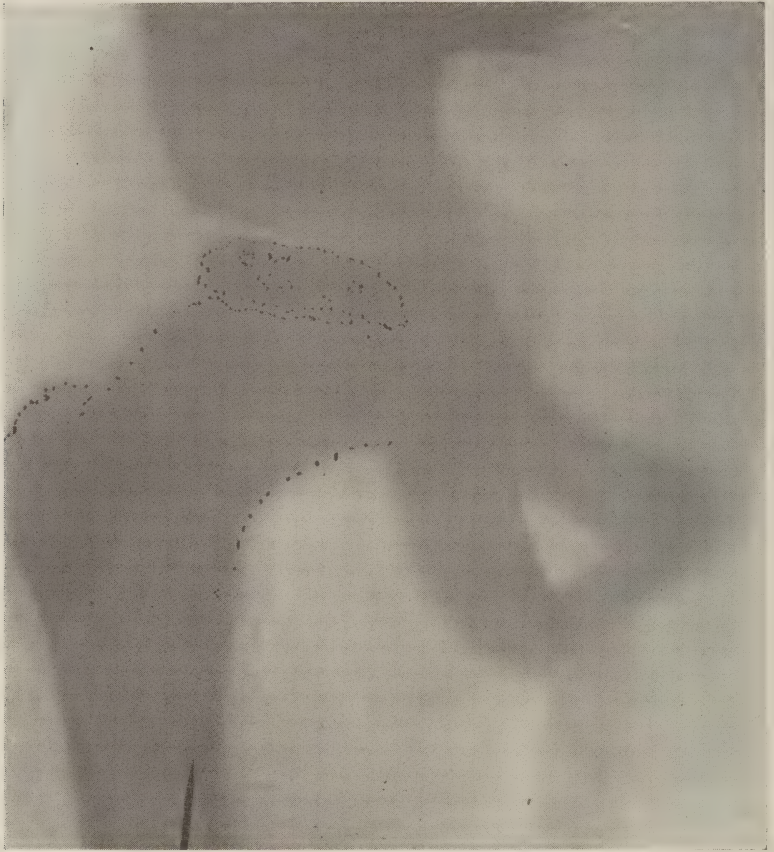


FIG. 131.—Legg's Disease. Note the irregularity in the epiphysis.

sis filled with granulation tissue and necrotic debris. The striking feature in his specimen is the dead bone. Many larger and smaller sequestra could be seen in the material scraped out of the cavity in the epiphysis. Under the microscope, the dead trabeculae could be made out.

SYMPTOMATOLOGY.—The thing for which a patient with Legg's disease almost invariably is brought for treatment is the limp. As a rule the limp is painless, though occasionally the child may complain of slight discomfort when he becomes tired.

On examination the extremity is seen to be in an indifferent attitude, or in one of slight adduction. Moderate muscular atrophy may be present but no real muscular spasm. The characteristic limitation of motion in this disease is in abduction. This is almost always decidedly compromised. Sometimes all abduction is abolished. Rotation as a rule is also restricted. Other motions may or may not be limited. In testing the range of motion a peculiar phenomenon usually can be brought out. When flexion is forced, the thigh goes into slight abduction, as it does in the second great type of arthritis.

Measurement shows the lower extremities to be of the same length, or possibly a shortening of about a centimetre in the affected extremity. The trochanter is prominent. In untreated cases the prominence of the trochanter increases, as does the shortening, and the limitation of abduction if this last has not been extreme from the start. The typical result of an untreated case is coxa vara, that is, a shortened, bent neck of the femur.

The disease runs its course in two or three years, with an active period of about one year. The constitution does not become affected, and the patient often runs and plays with his fellows.

The diagnosis may be made tentatively on the symptomatology, but it is clinched by the skiagram. This is characteristic.

The Röntgen rays show:

1. A flattening, broadening, and sometimes a lateral displacement, of the femoral epiphysis, with one or more divisions of it, and irregularity of ossification.



FIG. 132.—Legg's Disease. Note the irregularity in the epiphysis.

2. An irregularity or even a segmentation of the epiphysial disc.

3. Loss of bony structure of the neck, especially of its proximal and lateral part.

4. Irregularity of contour of the proximal border of the femoral neck.

5. Distortion of the head.

The first two are perhaps the most constant and im-

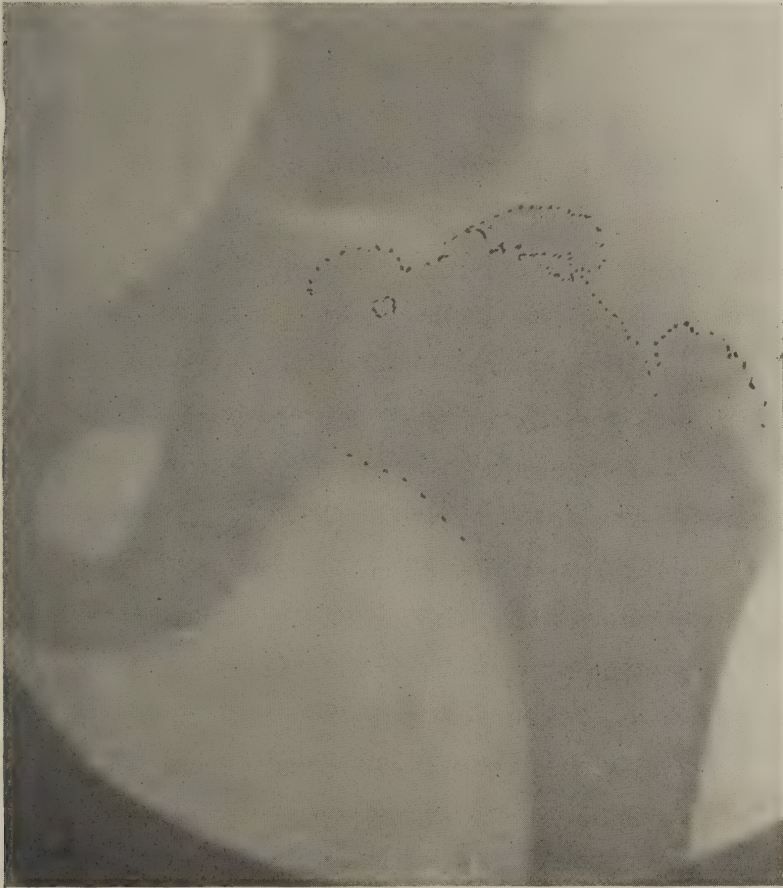


FIG. 133.—Legg's Disease. Note the irregularity in the epiphysis.


portant of these signs. In addition may be mentioned the occasional enlargement of the great trochanter. Occasionally also an irregularity in the outline of the acetabulum can be distinguished. What strikes the observer in

all this, is the marked disproportion between the extensive bone changes and the insignificant clinical symptoms.

Whatever its cause, evidently a softening of the femoral neck, especially that part of the neck in the region of the epiphysial line, is present. If the neck is exposed to weight bearing in the normal attitude, the epiphysis is displaced laterally more and more, and becomes more and more distorted, the neck bends and becomes thicker and shorter. This is the condition shown in the skiagram of the late stages of untreated cases, and it is probable that many cases of coxa vara in the adolescent are simply the end stages of an unrecognized previous Legg's disease.

TREATMENT

Opinions differ as to this. Phemister operated upon his patient, cleaning out the necrotic area in the epiphysis, and got a good result. Practically all other authorities advise conservative treatment. Perthes believes in massage and physical therapy, others recommend the traction brace to relieve the femoral neck from weight bearing. An excellent form of treatment is to abduct the hip to its extreme range, and put it up in that position in a short plaster spica. This forced abduction should be kept up with renewed spicas for a year or two, or until the Röntgen rays show a solidification of the femoral neck, and a more or less complete ossification of the epiphysis.



CHAPTER II

LOOSE BODIES IN THE JOINT

JOINT MOUSE

“OSTEOCHONDRITIS DISSECANANS”

WHEN we speak of a joint mouse we have a rather clear conception of our meaning in our mind, but when we attempt to give the term an exact definition, we find that the task is somewhat difficult. A joint mouse is a piece of bone or cartilage, or of bone and cartilage, loose in the joint, or attached to the capsule by a pedicle. However, not all pieces of loose cartilage are included in this category. As the result of tuberculosis in the marrow, larger or smaller fragments of cartilage are thrown off into the joint cavity, but these are not considered joint mice. Again in tabetic arthropathy, masses of bone and cartilage are separated, and lie in the joint, but these also are not joint mice. A joint fracture might occasion the presence of a piece of bone in the joint, but in the early stages of the trouble we should not call this a joint mouse. If, however, the fracture healed, the symptoms subsided, and the joint returned to an approximately normal condition, and if thereafter, the loose bone fragment should occasion trouble mechanically by its presence, we would speak of it as a joint mouse.

Islands of bone or cartilage are sometimes formed in the synovial membrane in the second great type of arthritis (*i.e.*, “arthritis deformans,” hypertrophic arthritis, degenerative arthritis, osteoarthritis, etc.), but they are not regarded as joint mice unless they constitute the main source of complaint, and overshadow the causal trouble,

then, whether they are loose or are still attached by a pedicle they are spoken of as joint mice. Otherwise they are simply recognized as bone and cartilage formations at operation. It is this factor of clinical importance that modifies our definition, and viewed in this light joint mice are of three kinds: first those which result from fracture,



FIG. 134.—Joint mice in the elbow.

second those which result from arthritis of the second great type, and third, the real “essential” joint mouse, formed in the articular end of a bone, usually the medial condyle of the femur.

As to the origin of the first class, there seems to be little discussion. If we believe that a fracture can cause the separation of a piece of bone and cartilage from the normal joint end of a bone, it is easy to understand that it will thereafter be loose in the joint cavity and constitute a

joint mouse. Such an occurrence, however, must be very rare, and while admitting the possibility, I confess that I have never seen a clear case of this kind.

The second class is more numerous. The islands of cartilage in the synovial membrane, occurring in the great second type of arthritis, may remain attached to the membrane, or may be loosened. It is likely that bone may be developed in them. Again it is possible that cartilage and bone may be broken off from the circumference of the articular ends of the bone, that is, from the new bone and cartilage that result from the lipping and exostoses in this form of arthritis. I have in my possession a stained slide from a piece of cartilage, about 5 m. in diameter, removed at hip resection. It was from one of these cases of arthritis of the hip, shows well-marked calcification, has a capsule of fibrous tissue and presumably was formed in the synovial membrane.



FIG. 135.—Two joint mice removed, exact size, from the elbow shown in Fig. 135. The lines on one show where the section from it was taken.

The third kind of joint mouse has occasioned much discussion. To account for its origin, two theories have been put forward. Koenig and his disciples maintain that a trauma to the end of the femur causes a localized necrosis in the bone end, and that a subsequent reactive inflammation separates this necrotic bone and cartilage, and sets it free. To this process Koenig gives the name "osteochondritis dissecans." Axhausen has attempted to prove this theory by wounding the articular cartilages of laboratory animals with an electric needle.¹ His views have

¹ AXHAUSEN: "Ueber einfache aseptische Knochen und Knorpelnekrose, Chondritis dissecans und Arthritis deformans," *Archiv. für Klinische Chirurgie*, 1912, xcix, 519.

obtained wide acceptance. Three sets of experiments carried out by Cowan and Ely,² throw grave doubt on the correctness of Axhausen's conclusions. In this connection it may be remarked that there can be no mechanical injury to a bone without a fracture.

Barth and his followers attribute these joint mice

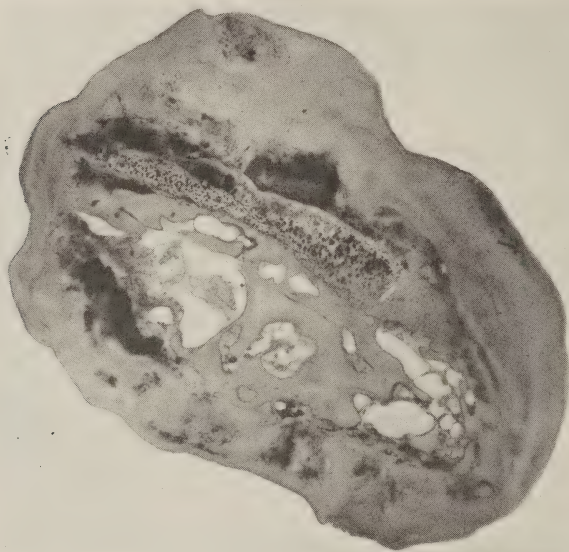


FIG. 136.—Photograph of a section of the larger of the mice shown in the preceding figure. X about 4 diameters.

directly to trauma, and deny the existence of any dissecting inflammation. They maintain, in other words, the fracture theory.

Several objections can be urged against these two theories.

In the first place, tuberculosis furnishes the best possible example of an osteochondritis dissecans, yet it never

²ELY AND COWAN: "Bone and Joint Studies I." Stanford University, Cal. Published by the University, 1916.

produces a typical joint mouse. It gives rise to an inflammation in the marrow which kills the bone trabeculae, and often throws a sequestrum of bone and pieces of cartilage into the joint. Examined under the microscope, these have no resemblance to a joint mouse.

By Koenig's explanation, a trauma *injures* the bone end, but exactly what could the injury be, that would cause this resulting dissecting inflammation? The soft tissues in the bone are beautifully protected from all injuries except fracture.

Against the fracture theory may be urged the fact that the injury which starts the trouble is not, as a rule, a severe one, and, as was said above, cases of demonstrated fracture with a distinct history, rarely are followed

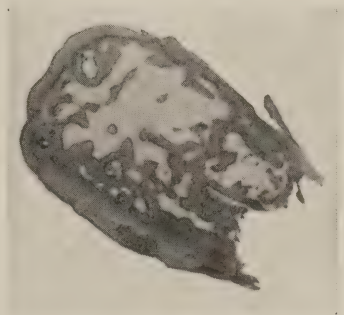


FIG. 137.—Low power photograph of the smaller of the joint mice. X about 4 diameters.

by these joint mice. Again, various writers have published cases of typical joint mouse in which the articular cartilage was intact over them, and the joint mouse lay held in its bed by the intact cartilage. A subcartilaginous fracture would be almost unthinkable.

There are three peculiar things about the joint mouse: 1st, its structure; 2nd, its location; 3rd, the age of the patient. It usually is found in young male adults or adolescents.

When one studies a joint mouse under the microscope, one is struck by the resemblance of its structure to that observed in foetal bones during the stage of intracartilaginous ossification. The resemblance is marked.

The location in the medial condyle has been mentioned.

The age at which these joint mice begin to give trouble, is that at which ossification in the lower epiphysis draws near completion, and the epiphysis joins to the shaft—about 20 years.

The “essential” joint mouse is always single, but the other variety may be multiple.

It seems then that neither of the best known theories of the origin of the true joint mouse is correct, and that the condition is due to an anomaly in the development of the distal epiphysis of the femur, possibly to an extra centre of ossification which fails to unite with the rest of the epiphysis.

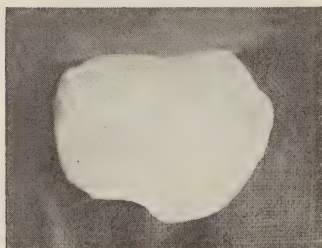


FIG. 138.—Loose body removed from joint, natural size.

SYMPTOMATOLOGY.—Loose bodies are far more frequently met with in the knee than in any other joint, and their symptomatology is markedly similar to that of a torn semilunar cartilage.

Indeed, by the very definition of a loose body, the torn semilunar might be included, but for the sake of clearness it is well to keep the two things separate in our minds. One is essentially from the bony structure, the other from the soft parts.

In the great majority of cases the history starts with a trauma, and, as a rule, the trauma is not great. The patient, in jumping, or falling, injures his knee. The joint swells, and exhibits all the signs of an acute traumatic arthritis. The arthritis subsides, and the joint usually returns to normal, and remains so until the body becomes pinched between the joint ends. The joint may lock, and the patient fall to the ground. Again, a simple sprain may result, a rupture of part of a ligament. The synovial

membrane is torn, and blood pours into the joint. A number of these attacks may weaken the joint, and give rise to a more or less chronic arthritis.

DIAGNOSIS.—Oftentimes these loose bodies can be felt through the skin. The patient may be able to locate them



FIG. 139.—Röntgenogram showing defect in surface of condyle of femur produced by the separation of the piece of cartilage.

himself. The greater number, those that consist partly of bone, can be detected with the Röntgen rays.

TREATMENT.—This consists exclusively of operative removal. After the body has been located, an incision is made through a convenient part of the joint capsule, and the body is caught with a pair of forceps and is removed. If there are others they should be removed also. Scrupu-

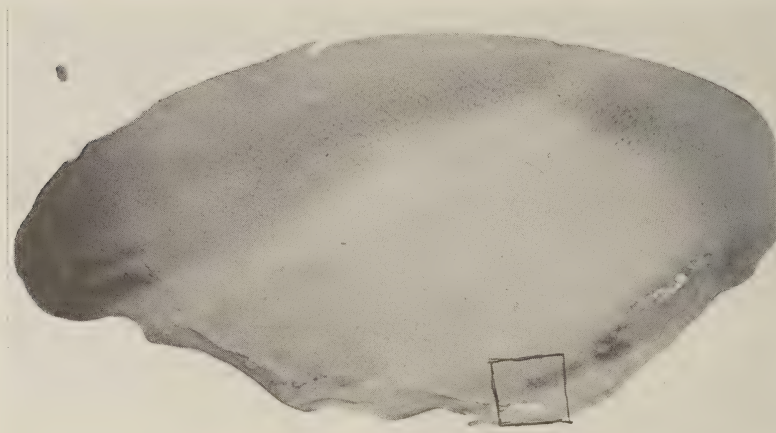


FIG. 140.—Typical joint mouse from medial condyle of the knee; photograph of stained slide of section from the joint mouse shown in Fig. 138. The marked square represents the area taken for the next illustration.

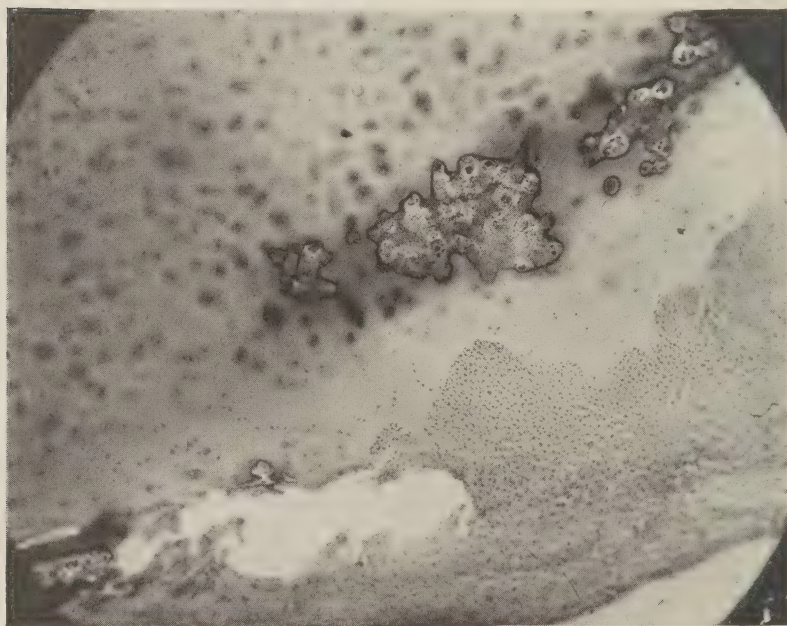


FIG. 141.—Low power photomicrograph of the marked portion of the preceding. The picture here presented strongly suggests intracartilaginous ossification.

lous care should be exercised in asepsis. The finger, gloved or ungloved, should not be inserted into the joint. It is often inserted, and of course the procedure gives one the sense of thoroughness, but it is unnecessary and dangerous. Suture of the capsule, and of the superficial tissues, completes the operation.

Sometimes the joint mouse will be found lying in its bed in the medial condyle of the femur, from which it never has departed. One must not forget at operation, those rare cases in which the intact articular cartilage holds the otherwise loose body in place. These cases show no sign at operation, of the joint mouse in spite of the fact that the X-rays have shown its presence. One tests out the cartilage by pressure, incising it where it yields.

CHAPTER III

KOEHLER'S DISEASE

DEFINITION.—A peculiar disease, or more properly, a peculiar condition, consisting in an abnormal shape, size and density of the tarsal navicular bone, first described by Koehler, of Wiesbaden, in 1908.

ÆTIOLOGY.—Boys are more often affected than girls in about the proportion of two to one. The age limits appear to be one and ten years. The great majority of cases are met with between the ages of three and seven, and five is the age at which the condition most often is seen.

Infection and trauma have been held responsible for Koehler's disease, but nothing that we know of the effects of trauma and infection upon bone indicates that either of them could cause the condition revealed by the Röntgen rays. The peculiar shape and consistency of a certain bone observed at a certain stage of development, which always disappears as growth proceeds, constitutes a strong argument that Koehler's disease is due to a developmental anomaly. Its occasional bilateral occurrence increases the strength of the argument.

SYMPTOMATOLOGY.—Either following an injury, or spontaneously, pain is felt over the navicular bone. This pain is made worse by exercise. Sensitiveness is also present, and with it may be associated swelling and redness. The patient limps, and may walk on the lateral border of the foot to save the medial part of it.

The symptoms persist for a longer or shorter time, and then disappear. They may last for a few weeks, or

for a few months. Complete recovery always takes place under any treatment.

The diagnosis is made by means of the Röntgen rays. These show the peculiar shape and consistency of the navicular bone. It is narrow, that is, the distance from its proximal border to its distal is diminished, but the gap between the talus and the cuneiform, indicates the presence of cartilage on each side of the bone nucleus. The bone itself is irregular in outline, and very dense in structure, so dense that the bone architecture, as ordinarily seen in the plate, is absent. The shadow is much darker than that of the neighboring bones.

TREATMENT.—This is largely a matter of indifference. If the foot is protected from injury and strain, the patient will probably be more comfortable.

CHAPTER IV

OSGOOD-SCHLATTER DISEASE

OSGOOD'S DISEASE, SCHLATTER'S DISEASE

SOMEWHAT akin to the preceding group, is a painful condition of the tubercle of the tibia, described first by Osgood,¹ and a few months later by Schlatter.² Its underlying cause is a peculiar formation of the proximal epiphysis of the tibia, but the symptoms probably owe their origin to trauma. Authorities differ as to the exact nature of the complaint, and it is necessary, in the first place, to exclude from the category cases of distinct fracture of the tibial tuberosity, though this injury produces a clinical picture hardly to be distinguished from that of Osgood-Schlatter disease.

If a large number of X-ray plates of the proximal end of the adolescent tibia be examined, the majority will show the epiphysis to have the shape of a fairly regular disc. A large proportion will show anteriorly a distal prolongation of the epiphysis in the form of a beak, or horn. This peculiar bone development is not to be regarded as at all abnormal in itself. It occurs too frequently for that, and its existence causes no symptoms. It is to be viewed simply as an irregularity, but as an irregularity which predisposes to injury. Sometimes the beak is quite regular, sometimes it is roughened or segmented, but its presence seems to predispose to this so-called disease.

¹ OSGOOD, ROBERT B.: "Lesions of the tibial tubercle occurring during adolescence." *Boston Med. and Surg. J.*, 1903, v. 148, 114.

² SCHLATTER, CARL: "Verletzungen der schnabelförmigen Fortsatzes der oberen Tibiaepiphyse." *Beit. zur. klin. Chir.*, 1903, v, 38, 874.

Most authorities agree that trauma stands in a causal relation to the symptoms, though some hold to the infectious theory, and some predicate a congenital tendency to general aberrant epiphysial development. Whether the trauma is direct upon the epiphysial beak, or indirect by

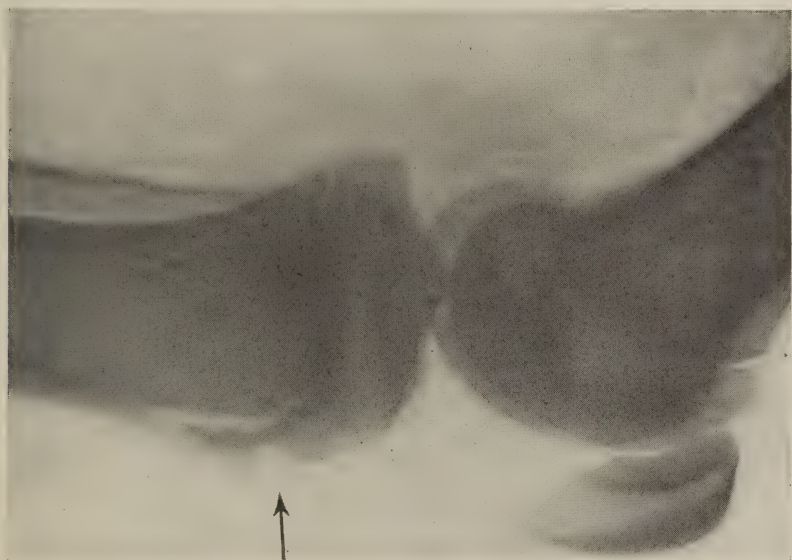


Fig. 142.—Osgood-Schlatter Disease.

pull of the quadriceps tendon, is still a matter of debate. Probably it can be either. In this connection, Heron's observation, that, when the beak is present, it serves for the attachment of the patellar tendon, is important. Strong contraction of the quadriceps muscle then would tend to loosen the epiphysis. This would result in a condition analogous to a chronic sprain. The situation of the beak, on the other hand, exposes it to direct violence.

Osgood-Schlatter disease is seen only in the young. Symptoms may present themselves apparently at any time, from the appearance of the ossific centre in the proximal

epiphysis, until union of the epiphysis with the shaft. Seven to eighteen years may be said to be about the limits.



FIG. 143.—Osgood-Schlatter Disease.

Authorities differ markedly as to the most vulnerable age. Some find the condition oftener in the right leg, some in the left. As with Legg's disease, joint mouse, and Koehler's disease, boys are afflicted much more frequently than girls.

Often the peculiar beak is present on both tibiae, on one without symptoms. Röntgen pictures taken for other conditions about the knee sometimes disclose the beak as an accidental finding. Occasionally the continuity of the beak with the epiphysis is broken, and a separate centre for the tubercle replaces the beak.

SYMPTOMATOLOGY.

—The patient, usually a young boy, complains of more or less pain in the exact location of the tibial tubercle, and often gives a history of an injury of not severe degree. Examination shows moderate swelling, without fluctuation, or crepitation. The swelling, is extremely sensitive to pressure, and may be reddened, but never ecchymotic.

The diagnosis is made with the Röntgen rays. They show the peculiar bone development at the tibial tuberosity, usually in the form of a beak, and more or less notched and irregular.



FIG. 144.—Osgood-Schlatter Disease.

Various operations have been recommended for this affection, but they seem unnecessary. Massage or strapping with adhesive tape suffices for the milder cases. The more severe ones yield to immobilization with plaster of Paris.

ARTHRITIS FROM CONGENITAL ANOMALY OF THE PATELLA

Attention has been called by several writers to irregularities in the development of the patella as well as to the presence of a bipartite or supernumerary patella, and these irregularities have been assumed, with reason, to be the cause of attacks of synovitis in young adults, recurring after strenuous physical exertion. They are revealed by the Röntgen rays.

The treatment is that of any mild traumatic arthritis. No one has proposed as yet, any treatment of the causal anomaly. It seems to incapacitate a patient for hard physical exercise, such as marching.

GOUTY ARTHRITIS

The characteristic lesions of this form of arthritis are the necroses in the bone and cartilage, and the deposition of crystals of biurate of sodium in the bone, in the cartilage, and in the capsule. The arthritis itself, is probably the result of these gross primary changes, and is therefore essentially a traumatic arthritis.

The diagnosis is made on the chalky deposits in the region of the joints, especially in the region of the metatarsophalangeal joint of the great toe. The X-ray picture, with its punched out areas in the bone near the articular surface, is quite characteristic. In the fingers the disease, must be carefully distinguished from the second great type of arthritis, the so-called Heberden's nodes.

Possibly gout may be caused by some form of protozoon.

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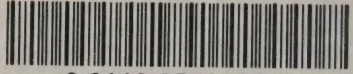
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